Trauma and dissociation in psychosis.

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Declaration

**This thesis has been composed by myself,
the work contained herein is my own
and it has not been submitted for any other degree
or professional qualification except as specified.**

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Abstract

In this thesis we attempt to address the complex issue of the relationship between trauma, dissociation, and psychosis. We start by providing a brief presentation of the background to this thesis, which is followed by an outline of the main clinical aspects and theories of psychosis. Subsequently, a broad evolutionary overview of trauma is given within which existing influential cognitive theories of PTSD are placed. Current models of dissociation are then reviewed and related to the view of trauma and traumatic stress reactions previously outlined, before providing an evaluative synthesis of the theoretical approaches and convergent conceptualisations of trauma, dissociation, and psychosis in order to disentangle some of the plausible processes underlying their relationship.

It was hypothesised that dissociation, occurring as a result of trauma (experience of psychosis), plays a key role in the formation and maintenance of psychotic symptoms, chiefly hallucinations and delusions. We used methods from experimental psychopathology to investigate the potential role played by dissociative processes in the disruption of the cognitive processes of attention and memory for trauma-related, positive and neutral information in two groups of participants: 30 individuals with psychosis and 30 matched controls. In particular, we used self-report measures of symptomatology, recovery style, trauma-related symptoms, and dissociation, and

employed two experimental tasks. The first was specifically devised to assess attentional processes: a Directed Forgetting Stroop Task (DFST) performed under conditions of divided attention. The second task was a Word-Stem Completion Task (WSCT) on which we applied the process dissociation procedure (PDP; Jacoby, 1991) in order to estimate the relative contribution to dissociation of implicit and explicit memory.

As expected, our findings revealed that compared to controls the experimental group processed information preferentially in an implicit manner, and that this effect was predicted by levels of dissociation and trauma-related distress. Although enhanced unconscious memory was not specific to trauma-related material, it significantly contributed to the level of positive symptomatology when mediated by stress levels. In contrast, the contribution of recovery style in the maintenance of psychotic symptoms was not supported, although this may reflect a limitation of the self-report measures employed in our study. Contrary to what was hypothesised, we did not find a standard directed forgetting effect in our memory task or an advantage (less interference due to dissociation) in our task of divided attention.

Results are discussed in the light of the theoretical background, previous experimental literature, methodological limitations, and current models of trauma and dissociation.

CONTENTS

Chapter 1

Psychosis: clinical aspects and theories

"As an experience, madness is terrific…
and in its lava I still find most of the things I write about."

(Virginia Woolf, "Letter to Ethel Smyth, 22 June 1930")

1. 1. General introduction.

In recent years, increasing interest and attention have been paid to the relationships among psychological trauma, psychotic episodes, dissociative phenomena, and cognitive functions of attention and memory (e.g. Allen, Coyne & Console, 1997; DePrince & Freyd, 2001; Gershuny & Thayer, 1999; Morrison, Frame & Larkin, 2003; Siegel, 1996).

It is widely accepted that exposure to subjectively traumatic life events may lead to psychological distress and contribute to the development of psychopathology in some individuals (e.g. Brewin, Andrews & Valentine, 2000; Christopher, 2004).

The definition of what constitutes psychological trauma or traumatic life events varies across studies. Consequently, in reviewing the literature we shall necessarily include studies that have used heterogeneous criteria and methodologies in their investigations.

The prevalence rate of traumatic events in individuals with psychosis is particularly high, with studies reporting up to 98% of their clinical samples having experienced at least one traumatic event (e.g. child abuse, physical attack) in their lifetime (e.g. Mueser, Goodman, Trumbetta, Rosenberg, Osher, Vidaver, Auciello & Foy, 1998; Resnick, Bond & Mueser, 2003; Seedat, Stein, Oosthuizen, Emsley & Stein, 2003).

Moreover, and in addition to the presence of a trauma history as a *distal* contributory factor to the development of psychosis, several authors have also examined the traumatic nature of the development of an acute psychotic episode and the experience of residual symptoms (e.g. McGorry, Chanen, McCarthy, van Riel, McKenzie & Singh, 1991; Meyer, Taiminen, Vuori, Äijälä & Helenius, 1999; Shaw, McFarlane, Bookless & Air, 2002; Stampfer, 1990). Specifically, it has been argued that the occurrence of psychosis may be seen as a traumatic life event in itself, which in some individuals may lead to the development of post-psychotic post-traumatic stress disorder (PP-PTSD) symptoms and constitute a *proximal* contributory cause of poor prognosis and relapse.

Dissociative processes (e.g. depersonalisation, derealisation, emotional numbing, explicit - but not implicit - memory loss) are considered to be a functional defensive response during the experience of trauma which moderate the psychological impact of distressing events on an individual (e.g. Kihlstrom, 2001; van der Kolk, Pelcovitz, Roth, Mandel, McFarlane & Herman, 1996). However, peritraumatic dissociation and high trait dissociation have also been associated with an increase in PTSD symptoms following trauma (e.g. Murray, Ehlers, & Mayou, 2002; Ozer, Best, Lipsey, & Weiss, 2003; Shalev, Peri, Canetti, Schreiber, 1996).

The relationship between dissociation and psychotic symptoms is generally recognised (e.g. Allen & Coyne, 1995) as is its aetiology in traumatic experiences (e.g. Steinberg, 1995). Specifically, Allen and colleagues (1995; 1996; 1997) have suggested that dissociation (dissociative detachment, in particular) increases vulnerability to psychotic episodes in that "the anxiety-driven retreat into dissociative detachment undermines patients' moorings in reality and may render them more vulnerable to flashbacks associated with the activation of traumatic memory networks" (1996; p. 641).

Indeed, both clinical practice and research (e.g. Read & Argyle, 1999) indicate that delusions and/or hallucinations can often reflect the content of past trauma. Therefore, although dissociation may be an adaptive defence mechanism during the experience of trauma, it may also be maladaptive later on, by blurring the boundaries between inner vs. outer, past vs. present, and lead to impairment in reality testing, disorganised thinking and increased vulnerability to further psychotic episodes.

It has been noted that PTSD and psychotic symptoms contain some phenomenological similarities (e.g. Butler, Mueser, Sprock & Braff, 1996; Nayani & David, 1996). For instance, intrusive recollections or "flashbacks" characteristic of PTSD are often experienced in the form of visual, auditory, olfactory or tactile hallucinations, and are accompanied by paranoia; whereas, other symptoms such as increased arousal, hypervigilance, disturbed sleep, emotional numbing, detachment and derealisation, appear to be common to the two disorders.

As we shall see in more detail later in this thesis, from a theoretical point of view, some models of PTSD and psychosis appear to converge on their proposition regarding the basis and development of involuntary intrusions. Specifically, recent cognitive theories of PTSD (Brewin, Dalgleish & Joseph, 1996; Ehlers & Clark, 2000) view intrusions as due to the representational format of trauma memories which lack spatial and temporal context: "the trauma memory is poorly elaborated and inadequately integrated into its context in time, place, subsequent and previous information and other autobiographical memories" (Ehlers & Clark, 2000; p. 325). It is argued that poor contextual integration leaves an individual vulnerable to the involuntary activation of trauma-related memories in response to situational reminders of the trauma (e.g. sights, sounds, smells). Brewin et al. (1996) refer to this process as reflecting the operation of a "situationally accessible memory" (SAM) system.

On the other hand, Hemsley's (1996) cognitive model of schizophrenia proposes that individuals with psychosis exhibit a weakened ability to integrate information within a temporal and spatial context, which results in the occurrence of hallucinations and delusions. This deficit is deemed to be generally present in individuals with psychosis, although it may be exacerbated by the experience of trauma.

Some recent evidence in support of this information-processing view of the development of PTSD intrusions and psychotic symptoms exists. Specifically, temporal and contextual deficits have been found in individuals with psychosis, schizotypal personality disorder, and individuals scoring high on schizotypy (i.e. psychosis proneness) (Barch, Mitropoulou, Harvey, New, Silverman & Siever, 2004; Elvevåg, Brown, McCormack, Vousden & Goldberg, 2004; Peters, Nunn, Pickering & Hemsley,

2002; Steel, Hemsley & Pickering, 2002). Additionally, high schizotypy, dissociation, and experimental manipulation that facilitates encoding into the SAM system have been found to significantly predict the increase of traumatic intrusions in healthy individuals following the viewing of a trauma film (Holmes, Brewin & Hennessy, 2004; Holmes & Steel, 2004).

These observations raise important questions about the way the appraisal of traumatic life events, dissociative processes and the representational format of trauma memories may contribute to the development and maintenance of psychotic symptoms.

Dissociative processes, in particular, have been invoked for their potential to interfere with the encoding of trauma-related information, and for their possible mediating role in the relationship between trauma and PTSD and psychotic symptomatology (e.g. Allen, Coyne & Console, 1997; Brewin & Holmes, 2003; Morrison et al., 2003; van der Kolk et al., 1996).

From a cognitive perspective, dissociation has been conceptualised as a disruption and separation in the usually integrated functions of consciousness, identity, perception of the environment and memory (e.g. American Psychiatric Association, 1994; Siegel, 1996). However, despite its clinical importance, the cognitive underpinnings of dissociation are not well understood.

Several studies have used various experimental tasks (e.g. emotional Stroop, directed forgetting task, free recall, recognition) in order to examine the role of attention and conscious and unconscious memory during dissociative experiences (e.g. DePrince & Freyd, 1999; Elzinga, Phaf, Ardon & van Dyck, 2003; McNally, Clancy & Schacter,

2001; Zoellner, Sacks & Foa, 2003), however, conflicting results have been reported. For instance, Zoellner et al. (2003) have found no evidence of an avoidant or an intrusive encoding style for trauma-related material in PTSD clients even after the use of a "dissociation" mood induction procedure; whereas, DePrince and Freyd (2001) have reported that healthy participants with high scores of dissociation tend to recall fewer trauma and more neutral words when the experimental task involves divided attention, highlighting the potentially important role played by the attentional context during dissociative processes (a more comprehensive review of relevant studies is reported in Chapter 3, Section 3. 2.).

Inconsistencies in results appear to be due mainly to different population samples being studied (e.g. undergraduate students, individuals with PTSD, dissociative identity disorder (DID), history of abuse), and different methodologies being employed.

To date, and to the best of our knowledge, no study has experimentally investigated dissociative phenomena in individuals with a history of psychosis. Additionally, no study has included tests of attention and memory (implicit and explicit) in a single experiment that is methodologically sound. Therefore, no conclusions can be drawn with regards to the cognitive mechanisms of dissociation and their potential impact on the alteration of attentional and memory functioning for trauma-related information, which could be explained in terms of attention deficit/avoidance, disrupted encoding, or retrieval inhibition. Furthermore, no firm deductions can be made about the possible role of dissociation in the vulnerability, onset and maintenance of psychotic (as well as other) disorders.

In this context, the research project described in this thesis had a twofold purpose. The first aim was to investigate the pathway of trauma-related information processing in individuals who had experienced at least one psychotic episode, who presented with residual symptoms, and who were consequently hypothesised to be prone to dissociation, in comparison with a healthy control group. This would allow us to examine experimentally some of the cognitive processes underlying the hypothesis introduced above that dissociation, occurring as a result of trauma, might render individuals vulnerable to experiencing repeated psychotic episodes (e.g. Allen et al., 1997).

To this aim, two experimental tasks were used to evaluate the involvement of attentional and mnemonic processes occurring, as hypothesised, during dissociative phenomena. The first task was an Emotional Stroop Task merged with a Directed Forgetting Task (list method), performed under conditions of divided attention. Stimuli included trauma-related, positive and categorised neutral words. Following the study phase, by applying the process dissociation procedure (PDP; Jacoby, 1991) to a subsequently administered Word-Stem Completion Task, we were able to measure the relative contribution to dissociation of implicit and explicit memory for trauma-related compared to positive and neutral information.

According to the literature reviewed thus far, we expected to find a disruption in information processing specific to trauma-related material, which would be manifest in an attentional bias and, most importantly, impaired explicit (but not implicit) memory performance. The preservation of implicit memory, typically observed in dissociative

disorders (e.g. Kihlstrom, 2001), would indicate the presence of trauma-related material which is beyond conscious control and that might render an individual vulnerable to traumatic/psychotic intrusions due to its involuntary activation in response to situational triggers (i.e. word-stems, in our case).

Although we recognise the presence of a cumulative effect in terms of the contribution of *distal* and *proximal* traumatic life events to an individual's propensity to dissociate and his/her vulnerability to psychopathology, in this thesis we focused on examining the potential increased vulnerability brought about by more recent distressing experiences. Explicitly, in line with the view outlined above (e.g. McGorry et al., 1991; Shaw et al., 2002), we adopted the stance that the experience of psychosis is in itself a traumatic life event which is likely to cause trauma-related symptoms and that requires an individual to adapt and make major readjustments in relation to his/her life roles and goals. Consequently, the second objective of this thesis was to explore the relationship amongst severity of symptomatology, current levels of the psychological impact of trauma (i.e. experience of psychosis), dissociation, and recovery style – i.e. *integration* vs. *sealing over* (e.g. McGlashan, 1987) – in our clinical sample by using self-report questionnaires, and also to evaluate their relative influence on the participants' performance in the experimental tasks.

However, before we begin to report on the empirical work carried out, we shall dedicate the remaining part of this chapter to the introduction of the main clinical aspects and psychological theories of psychosis. Then, in Chapter 2, we shall present some of the

current issues related to the concepts of trauma, dissociative phenomena, and examine the relationship between trauma and dissociation and their particular relevance to psychosis.

1. 2. Psychosis: definition and clinical aspects.

The term *psychosis* is conventionally used to describe the most severe psychiatric disorders. Traditionally, a distinction has been made between psychosis and *neurosis*, whereby psychosis has been understood as a condition with organic aetiology in which the individual affected is thought to have lost touch with the objective reality, whereas neurosis has been seen as a condition with psychological aetiology in which contact with reality is preserved (e.g. Beer, 1996). Additionally, since for most psychiatric disorders there is a lack of clear biological correlates, two different types of psychotic disorders have been proposed: *organic psychoses*, for which the origin of symptoms can be identified as due to organic damage in specific brain loci, and *functional psychoses*, for which an obvious cause is unknown but it is assumed to be organic, in whole or in part. Therefore, traditional approaches to diagnostic classifications of mental health problems assume a hierarchical structure with varying degrees of supposed brain dysfunction: organic psychoses, functional psychoses, and neurotic disorders at the end of the hierarchy, since they are assumed to be largely non-organic in their aetiology. This traditional view changed somewhat with the publication of the DSM-III (American

Psychiatric Association, 1980), which abandoned the major categorical distinction between psychosis and neurosis.

To date, functional psychoses have been conventionally divided into *non-affective* (e.g. schizophrenia) and *affective* psychoses to indicate the absence or presence respectively of a characteristic emotional tone of the symptoms presented (e.g. depression, mania).

Since the 1960s, when research into the effects of different social environments in psychiatric institutions was carried out (e.g. Wing & Brown, 1961), it has become conventional to separate symptoms of schizophrenia into two syndromes signifying functional distortions or excesses (*positive*) and deficits (*negative*). Positive symptoms include:

- *delusions*: erroneous beliefs held in spite of evidence to the contrary, that are not accepted by other members of the individual's culture (or subculture);
- *hallucinations*: sensory experiences in the absence of any stimulation from the environment;
- *thought disorder*: problems in the organisation of ideas and speech;
- *disorganised behaviour*: difficulties in self-monitoring, bizarre or catatonic behaviour;

whereas, negative symptoms include:

- *apathy* or *avolition*: lack of interest in initiating goal-directed behaviour;
- *alogia*: poverty of content and reduced speech;
- *blunted affect*: restrictions in the range and intensity of emotional expression;
- *withdrawal*: social and emotional.

This set of symptoms has been incorporated into current diagnostic classification systems, and the presence of positive symptoms in particular has become for most clinicians a strong indicator that an individual is suffering from a psychotic episode and should be given a related diagnosis (e.g. schizophrenia) despite this not carrying necessarily any particular theoretical or prognostic value.

In fact, notwithstanding the identification of the above symptoms, there is evidence that psychosis exists on a continuum with normal and qualitatively similar experiences that occur in the general population (e.g. Kendler, Gallagher, Abelson & Kessler, 1996; Myin-Germeys, Krabbendam & van Os, 2003). For example, van Os, Hanssen, Bijl & Ravelli (2000) found that 17.5% of a random sample of 7076 individuals reported having experienced psychotic-like symptoms (i.e. high *schizotypy*, or psychosis proneness) and could be placed on a continuum between the non-clinical and clinical populations with 2% of these receiving a formal diagnosis of non-affective psychosis.

Moreover, it is well known that in conditions of extreme sensory deprivation (Leff, 1968), solitary confinement (Grassian, 1983), sleep deprivation (Babkoff, Sing, Thorne, Genser & Hegge, 1989), bereavement (Grimby, 1993) and hostage situations (Siegel, 1984) individuals can also experience hallucinations and delusions.

Therefore it would appear that a view of psychosis as continuous with manifestations ranging from psychotic-like experiences to psychotic disorders may be preferable to a model of discontinuity between normal and abnormal. It is more likely that, as it is the case with "neurotic disorders", clinical levels are reached once symptoms interfere significantly with the individual's biopsychosocial functioning.

The definition of particular criteria that might characterise the passage from normal to abnormal ranges of psychotic experiences has undergone several developments over the last fifty years, and today the DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition – Text Revision; American Psychiatric Association, 2000), and the ICD-10 (International Classification of Diseases – Tenth Edition; World Health Organization, 1992) are the most widely used diagnostic classification systems of mental disorders. They are both categorical in their classification and provide equivalent, although not identical, lists of symptoms grouped into separate types (or sub-types) of psychotic disorders. Table 1. 1. below, gives an outline of the types of psychotic disorders reported in DSM-IV-TR and ICD-10.

Table 1. 1. DSM-IV-TR and ICD-10 classification of psychotic disorders.

DSM-IV-TR	ICD-10
Schizophrenia	Schizophrenia
Schizophreniform disorder	Schizotypal disorder
Schizoaffective disorder	Schizoaffective disorder
Delusional disorder	Persistent delusional disorder
Brief psychotic disorder	Acute and transient psychotic disorders
Shared psychotic disorder	Induced delusional disorders
Substance-induced psychotic disorder	Other non-organic psychotic disorders
Psychotic disorder due to a medical condition	Unspecified non-organic psychosis
Psychotic disorder not otherwise specified	

Both these systems provide inclusion and exclusion criteria for diagnosing schizophrenia and other psychotic disorders. However, the proposed diagnostic concepts are to be regarded as provisional constructs (purely describing observable phenomenology) intended to foster the need for international communication and research on psychosis,

rather than categories reflecting any correspondence with specific underlying processes or aetiology.

A recent review of epidemiological studies (Jablensky, 2003) cites an overall worldwide prevalence of schizophrenia in the range of 1.4-4.6 per 1000 population at risk, whereas the incidence rate ranges from 0.17 to 0.54 per 1000 population per year. The typical age of onset is between late adolescence and mid 20s for males and late 20s to early 30s for females, although for the latter group there seems to be another increase in incidence after the age of 40, when the male-female ratio becomes inverted.

Different hypotheses have been put forward to try and explain the reasons for these gender differences. These have varied from the supposed protective effect of oestrogen in reducing the sensitivity of D_2 dopamine receptors (Häfner, an der Heiden, Behrens, Gattaz, Hambrecht, Löffler, Maurer, Munk-Jorgensen, Nowotny, Riecher-Kössler & Stein, 1998), to gender differences in premorbid levels of psychosocial functioning, with males showing a significantly higher frequency of socially adverse behaviour (e.g. alcohol and drug abuse, aggressive behaviour, self-neglect) and lower social adaptiveness at the early stages of illness than their females counterparts (Häfner, Maurer, Löffler, an der Heiden, Hambrecht & Schultze-Lutter, 2003).

Although after the age of 50 there are relatively few new cases of schizophrenia in either gender, late onset schizophrenia appears to be more common in females with a preponderance ranging from 66 to 91% (Howard & Jeste, 2003).

Since late onset schizophrenia appears to be characterised by a relative lack of thought disorder, fewer negative symptoms, and generally a more favourable course, it has been

hypothesised that the disorder represents a neurodegenerative process as opposed to a neurodevelopmental one in early onset (e.g. Tune & Salzman, 2003). Consequently, an early emergence of schizophrenia might be accompanied by a worse prognosis because of the adverse effect on the biological and psychosocial development in young people, whereas, individuals with late onset may already have acquired more adaptive skills when their illness strikes.

Most of the course and outcome studies of schizophrenia suggest that the course is variable and indicate a number of factors associated with a better outcome such as, good premorbid adjustment, acute and late onset, female gender, good insight, higher socioeconomic status, ethnicity (i.e. white), geographical region (i.e. developing country or rural setting), no family history of psychosis, level of expressed emotion (EE) within the family (i.e. low frequency of hostility, critical comments and emotional over-involvement) adherence to antipsychotic medication, good level of inter-episode functioning with minimal residual symptoms, early intervention (e.g. Zuckerman, 1999).

Given the large heterogeneity in presentation of individuals with psychosis in terms of different symptom profiles, age and type of onset (acute vs. insidious), variable response to available treatments, unpredictable clinical course, and lack of a relationship between diagnoses and aetiology, the validity of the classification systems outlined above have been seriously questioned (e.g. Bentall, 2003). Both the DSM-IV-TR and ICD-10, while providing the basis for effective communication among professionals, appear to create a false impression of the existence of discrete taxonomic entities, by imposing a

categorical framework and complex arbitrary algorithms on what are in fact overlapping phenomena. Doubts about the stability and reliability of the proposed diagnostic categories have also been raised (e.g. Arndt, Andreasen, Flaum, Miller & Nopoulos, 1995; Chen, Swann & Burt, 1996; Fenton & McGlashan, 1991) and despite any efforts in improving reliability, this in itself does not guarantee better construct validity.

Another fundamental problem with the current diagnostic systems regards the basic dichotomising of affective and non-affective psychoses. A diagnosis of psychosis normally takes precedence over other types of disorders, however, in most cases emotional disturbance (anxiety and depression in particular) precede, accompany, and follow a psychotic episode (e.g. Birchwood, 2003; Freeman & Garety, 2003). Kendler et al. (1996) found that individuals with non-affective psychoses had a lifetime prevalence of 73.4% for mood disorders and of 71.4% for anxiety disorders. As a result, the "paradox" of experience of emotions in non-affective psychoses, an issue neglected for a long time, is recently becoming the focus of attention for some researchers (e.g. Blanchard, Mueser & Bellack, 1998; Myin-Germeys, Delespaul & deVries, 2000). For instance, a recent study that looked at the experience of basic emotions in individuals with a diagnosis of schizophrenia with and without affective symptoms found that all patients tended to feel negative emotions such as fear, disgust, guilt, shame and anger more often than a healthy control group, but also that only patients with affective symptoms felt the positive emotions of joy and interest less often than controls; however, there was no significant difference between this latter group and patients without affective negative symptoms, which shows a full range (positive and negative)

of emotional experience in individuals with a diagnosis of chronic schizophrenia (Suslow, Roestel, Ohrmann & Arolt, 2003). In terms of the diagnostic value of the DSM-IV-TR and ICD-10 categories of affective and non-affective psychosis, some evidence exists that a high rate of affective symptoms is equally common in patients with either diagnosis, showing that the overlap between the two syndromes is the rule rather than the exception (van Os, Gilvarry, Bale, van Horn, Tattan, White & Murray, 2000). Of course, both classification systems have attempted to overcome this impasse by incorporating schizophrenia and affective disorders into the "intermediate" category of schizoaffective disorder.

A related question is the distinction between schizophrenia and bipolar disorder. The latter is described in DSM-IV-TR and ICD-10 as a disorder characterised by a pattern of recurrent affective episodes which include depressive and manic phases with intervening euthymic periods. However, despite this classic conceptualisation of bipolar disorder, many patients do not return to "baseline function" in between episodes and, during a psychotic episode, schizophrenia and bipolar disorder may be indistinguishable from each other, which alerts us of the possibility that the two may not be separate disorders (e.g. Adler & Strakowski, 2003, Bentall & Kinderman, 1999).

A different way of addressing this issue, rather than resorting to mixed categories, has been that of adopting a dimensional approach. Dimensional models attempt to define groups of symptoms that tend to co-occur through statistical techniques such as factor analysis. The resulting identified dimensions may then overlap within the same

individual who can also move along them over time. Several investigators (e.g. Grube, Bilder, Goldman, 1998; Ratakonda, Gorman, Yale & Amador, 1998) have identified three main independent dimensions underlying psychotic disorders: *a*) *reality distortion syndrome* (i.e. florid delusions and hallucinations), *b*) *psychomotor poverty syndrome* (i.e. poverty of speech, blunted affect, apathy/avolition, anhedonia, social withdrawal, decreased sponteneity), and *c*) *disorganisation syndrome* (i.e. attentional impairment, thought disorder, bizarre behaviour, inappropriate affect). However, other studies have isolated slightly different and interrelated dimensions such as positive, negative and depressive symptoms (Stefanis, Hanssen, Smirnis, Avramopoulos, Evdokimidis, Stefanis, Verdoux & van Os, 2002), have found a different number of dimensions (e.g. Cuesta & Peralta, 2001), or have used a hierarchical approach (Serretti, Rietschel, Lattuada, Krauss, Schulze, Müller, Maier & Smeraldi, 2001) in an attempt to reconcile categorical and dimensional models of psychopathology.

Therefore, while current diagnostic categories appear to lack in validity and reliability, properties that are required for use both in research and in clinical practice, dimensional approaches seem to provide more flexibility in the description of psychotic phenomena in terms of which symptom dimensions are present and their degree of severity.

We shall now turn to examine how the presence of psychotic phenomena has been explained by theorists belonging to different schools of thought. Although it is beyond the remit of this thesis to provide a comprehensive review of every theory of psychosis, at this point it is useful to introduce briefly some of the most influential psychological

accounts of psychotic disorders that are of particular relevance for our study. Specifically, a general overview of the neuropsychological, cognitive and psychodynamic approaches will now be considered in turn, since they will aid our understanding of psychotic phenomena as a composite of the disruption/distortion of cognitive processes and defence mechanisms. Finally, a summary of this chapter will be given.

1. 3. Theoretical approaches to psychotic disorders.

1. 3. 1. Neuropsychological perspective.

Neuropsychological models integrate neurological and cognitive explanations of psychosis. Such models are based on the assumption that the brain works somewhat like a computer in the way it processes information, by a number of modular processes working in parallel (e.g. Shallice, 1988). Generally, these models attempt to provide an understanding of how cognitive impairment, based on possible neurological damage, gives rise to psychotic experiences. Research has shown a number of cognitive neuropsychological deficits in people with psychosis. For example, people who meet diagnostic criteria for schizophrenia appear to function with IQs which are on average 10 points lower than premorbid IQ estimates (Nelson, Pantelis, Barnes, Thraser & Bodger, 1994). In terms of more specific cognitive deficits, individuals with schizophrenia have shown to have attentional problems (e.g. slower reaction times,

difficulty maintaining vigilance), episodic and working memory difficulties, and deficits in executive function irrespective of IQ scores (e.g. Aleman, Hijman, de Haan & Kahn, 1999; Goldberg, David & Gold, 2003; Weickert, Goldberg, Gold, Bigelow, Egan & Weinberger, 2000).

Several models have emerged that have tried to interpret neurocognitive deficits in a unitary fashion so as to identify a single deficit that might underpin symptoms of schizophrenia. A recent model suggests that the fundamental deficit in schizophrenia is a disruption of the fluid co-ordination of mental activity, referred to as *cognitive dysmetria* (Andreasen, 1999). The disruption in the synchrony of mental activity would manifest itself in difficulties in behaviour, cognition and emotion. Essentially, schizophrenia is seen as a neurodevelopmental cognitive disorder with a complex aetiology (interaction of genetics and environment) that results in abnormalities of brain development. These abnormalities (from conception to early adulthood) lead to the disruption of anatomic and functional connectivity in the brain which causes cognitive dysmetria and, consequently, positive and negative symptoms of schizophrenia. A similar neurodevelopmental model has been proposed also by Weinberger (1987; Weinberger & Marenco, 2003).

Frith (1987; 1992) proposed that underlying the symptoms of schizophrenia is a disorder of consciousness or self-awareness. It is argued that for people with schizophrenia, preconscious processes (e.g. the selection of the appropriate interpretation and response to stimuli) are frequently conscious, so that, for example auditory hallucinations are seen

as an awareness of incorrect preconscious interpretations of stimuli. Such a disorder impairs an individual's ability to think using metarepresentations or metacognitions (i.e. higher order abstract concepts) and causes three types of disorders characteristic of schizophrenia: *a*) *willed intention*, or the inability to generate spontaneous or willed actions, which results in inappropriate or poverty of action; *b*) *self-monitoring*, or inability to monitor willed intentions, which results in auditory hallucinations, delusions of control or thought insertion; *c*) *monitoring the intentions of others*, or the inability to understand the meaning of social situations, which results in paranoid delusions. Frith (1992) suggests that dysfunctions in the neuronal pathways between the septohippocampal system and the prefrontal cortex, as well as dopamine dysregulation in these areas of the brain, may be implicated in the disruption of consciousness.

An alternative but similar model has been proposed by Hemsley (1987; 1996). According to this model, individuals with schizophrenia exhibit a weakened influence of spatial and temporal regularities of past experience on the processing of current perception. In particular, it is argued that there is a disturbance of the moment by moment integration of stored memories with current sensory input, which results in information not being integrated within a temporal and spatial context.

Consequently, in schizophrenia an individual's experience of the sensory environment is affected by impairment in the rapid and automatic assessment of its significance. In other words, non-relevant features of the social environment are experienced as personally relevant, and thoughts and memories which are irrelevant to ongoing tasks are nonetheless assessed and perceived as alien and attributed to an external source. This

dysfunction in monitoring processes can therefore lead to individuals experiencing symptoms such as hallucinations and delusional ideas of reference. Some empirical support for this overly rapid assessment in individuals with schizophrenia comes from a study on probabilistic reasoning that found a tendency in this group to jump to firmly held conclusions on the basis of little evidence (Garety, Hemsley & Wessely, 1991). Again, disregulation of dopamine in the septohippocampal neuronal pathways has been proposed as the source of the deficit (Gray, Feldon, Rawlins, Hemsley & Smith, 1990).

Other deficits in information processing, particularly attention (Braff, 1993) and working memory (Goldman-Rakic, 1994), have been pointed out as being the cause of symptomatology in patients with schizophrenia. Hence, individuals affected by the disorder would have difficulties in focusing their attention (i.e. inhibiting it from irrelevant stimuli and allocating it to relevant tasks) thus feeling overwhelmed, and in organising their thoughts and behaviours as a result of an impaired memory system; both types of dysfunctions are regarded as being possibly due to disrupted prefrontal cortex circuitries.

All of these neuropsychological models appear to elevate one particular cognitive deficit and deem it to be fundamentally responsible for more extensive deficits in information processing and the resulting manifestation of several psychotic symptoms. Thus, different "essential" cognitive deficits have been put forward and corresponding neurological pathways have been proposed as the "solid" basis for these hypothetical models, most of which await empirical verification.

A different approach to the rather reductionistic "one explains it all" method, has been that of studying specific psychotic symptoms (e.g. Costello, 1993). This symptom-based approach is recognised as being the most useful and promising in terms of advancement of knowledge given the heterogeneity of psychotic manifestations. This type of investigation has led to the development of functional cognitive models of psychotic symptoms, reviewed next.

1. 3. 2. Cognitive perspective.

Cognitive models of psychosis are based on the assumption that psychotic symptoms do not necessarily form a syndrome which reflects the presence of an underlying disorder; rather, they offer discrete explanations and therapeutic approaches for individual symptoms which are seen as being on a continuum with normal experiences (e.g. Haddock & Slade, 1996). During the last 20 years or so, most of the advances have taken place in the development of models of positive symptoms, delusions and hallucinations in particular.

Maher (1988) suggested that delusions are simply the result of normal explanations of anomalous experiences. Once formed, delusional beliefs are then maintained by normal cognitive processes such as "self-fulfilling prophecy" (i.e. things turning out just as one expected because one behaves in a manner that optimises those very outcomes) and the experience of relief from puzzlement. Although this model provides a plausible explanation of beliefs formation and maintenance when anomalous experiences occur, it does not take into account vulnerability factors (i.e. why should individuals have

abnormal experiences in the first place) and the role of reasoning and emotional biases (e.g. Garety et al., 1991; Kaney & Bentall, 1992).

Another early model (Bannister, 1983) proposed that delusions can be seen as metaphors reflecting the rational description of bizarre or confusing experiences, and that as such, themes could be identified revealing an individual's underlying difficulties and concerns. While Bannister (1983) argued that the reason for using unsignalled metaphors might be that the person is distrustful of others and prefers to hide his/her real troubling thoughts and feelings, others have argued that the presence of cognitive deficits would interfere with an individual's capacity to form clear ideas and thoughts, which might result in communication based on what would appear to be unsignalled metaphors (Fowler, Garety & Kuipers, 1995).

More recently, Garety and Hemsley (1994) have examined the role of reasoning biases in the formation and maintenance of delusional beliefs. The authors have found evidence of two apparently contradictory biases in deluded individuals: a tendency to make less use of past learned regularities accompanied by excessive reliance on current information, and a tendency to rely excessively on prior expectations when processing new information. These two judgement styles have been proposed as reflecting two sequential stages in their model of delusions: formation and maintenance respectively. The authors have suggested that excessive reliance on current information leads to the formation of delusions; these then generate strong expectations and are maintained by a bias towards belief congruent information.

Research on attributional processes in individuals with delusions has led some investigators to propose that persecutory delusions have a defensive function (e.g. Bentall, 1994; Kinderman & Bentall, 1997; Lyon, Kaney & Bentall, 1994). These authors have found that individuals with persecutory delusions tend to attribute positive events to internal and negative events to external causes (other people rather than situations). They also show a specific attentional bias for threatening information and information relevant to the self-concept. Bentall and colleagues have suggested that deluded individuals have a negative self-schema and low self-esteem which are experienced as a discrepancy between the individual's ideal and actual selves. Consequently, delusions have a defensive function for these individuals who have an externalising bias for negative events in order to prevent thoughts and feelings about their low-self esteem reaching consciousness.

Trower and Chadwick (1995) have placed this conceptualisation of persecutory delusions as compensatory beliefs more explicitly within an interpersonal context suggesting that the source of threat to the self is an interpersonal negative evaluation. They have also extended this model to include punishment paranoia, characterised by the opposite attributional style (i.e. individuals attribute positive events to external and negative events to internal causes). However, since in punishment paranoia the individual believes that they are blameworthy and deserve punishing by others, its defensive function is less clear than in persecutory paranoia.

Attributional approaches to motivation and emotion (Weiner, 1986) have also been used to conceptualise hallucinations (auditory in particular) as a tendency to misattribute

internal events (e.g. one's own thoughts) to an external source (Bentall, 1990). This cognitive model of hallucinations is based on evidence from early studies that have shown the co-occurrence of auditory hallucinations with sub-vocalisations or movements of the speech musculature (Gould, 1950), and that have found that the occurrence of hallucinations can be inhibited by giving individuals verbal tasks that block sub-vocalisations (Margo, Hemsley & Slade, 1981). Bentall (1990) has argued that this attributional bias may be influenced by the presence of stress induced arousal, by the individual's beliefs and expectations about the self, the world and the future, and by the negative reinforcement (i.e. reduction of anxiety) that the process of misattribution will generate (i.e. avoidance of an aversive cognitive event: negative thoughts about the self).

A similar account has been given by Morrison, Haddock and Tarrier (1995). The authors have argued that auditory hallucinations arise from an individual's misattribution of ego-dystonic (i.e. incompatible with one's beliefs) thoughts to an external source. The presence of this process would explain both negative and positive auditory hallucinations in individuals with respectively positive and negative self-schema, in that it would reduce cognitive dissonance and distress. Indeed, the emotional valence of the auditory hallucinations has been found to have a direct effect on the bias in source monitoring (Morrison & Haddock, 1997), and on individuals' emotional and behavioural responses. Specifically, voices which were believed to be malevolent elicited negative affect and were resisted; whereas, voices believed to be benevolent were associated with positive affect and were engaged with (Chadwick & Birchwood, 1994; 1995). More

recently, Morrison (1998) has suggested some parallels between panic disorder and hallucinations and has argued that misinterpretations of auditory hallucinations are maintained by safety seeking behaviours, including hypervigilance. High levels of anxiety would also explain the misinterpretation of ambiguous external stimuli (e.g. noises or conversations in the next room) as threatening auditory hallucinations (e.g. Fowler & Morley, 1989; Haddock, Bentall & Slade, 1993).

The cognitive models reviewed above attempt an analysis of different ways in which people with psychosis might strive to make sense of adverse and unusual experiences. While this endeavour is useful in providing clues about the nature and the function of some types of hallucinations and delusional beliefs, and therapeutic working models for psychosis, their explanatory power appears to be somewhat limited to current possible precipitating and maintaining factors of the disorder. As such, these models lack the necessary emphasis on important developmental factors (e.g. early relationships, role transitions), which would add considerably to our understanding of psychotic symptoms. Further potential developmental contributory causes of psychosis are briefly reviewed next.

1. 3. 3. Psychodynamic perspective.

Psychodynamic theories of psychosis emerged with Freud's (1894/1962) proposal that the same basis for neurotic disorders (i.e. intra-psychic conflicts and defence mechanisms) also applied to psychosis. Despite his initial unitary approach, Freud

(1940/1964) gradually came to believe that because of the unique nature of psychosis, individuals were unable to establish the close interpersonal relationship essential for analytic work (i.e. form a transference relationship with the analyst). However, other psychoanalysts such as Fromm-Reichmann (1950) and Sullivan (1962) disagreed with this view and went on to adapt treatment techniques in order to be able to remain therapeutically engaged with people with psychosis. They suggested that the psychotic Ego is fragile and unable to deal with the stress of interpersonal challenges other than regressing to early childhood forms of communication. Hence, the aim of therapy was for the patients to gain insight into the role that the past had played in their current difficulties and to gradually learn adult forms of communication. In his historical review of the development of psychoanalytic ideas about psychosis, Frosh (1983) proposed that, ultimately, psychosis results from the loss of the capacity to test reality, the process of which hinges on adverse childhood experiences.

Several theorists (e.g. Bion, 1962; Klein, 1946/1986; Kohut, 1977; Winnicott, 1960) have contributed to the development of psychodynamic concepts on psychosis. In a nutshell, inadequacies in the interactions between a baby and its main caregiver (e.g. poor parenting, trauma), may lead to the development of a vulnerable Ego in the form of insecure sense of identity, poor self-esteem, insecure boundaries between self and others, and difficulties in relating to others. If a fragile sense of self is threatened by adverse life events, the individual will respond by activating an immature pattern of defence mechanisms characteristic of early childhood such as, splitting, projection, denial and distortion. Thus, for instance, at times of emotional difficulty, a vulnerable

self is likely to "split off" unbearable negative emotions and project them into the external world (e.g. somebody else), which will then become the source of "badness" or persecution. This defence will have the function of protecting the Ego from underlying negative feelings (e.g. anxiety, low self-esteem). Consequently, psychosis is seen as the result of the vulnerability of the Ego coupled with the vulnerability to early defence processes, which distort and impair the individual's perception and reality testing.

Psychodynamic theories also recognise the validity of genetic predisposition to psychosis (e.g. Robbins, 1993), so that the vulnerability to immature defence mechanisms is seen as the product of the interaction between genetic vulnerability and the experience of poor parenting, which result in unfavourable developmental pathways.

As a whole, the psychodynamic approach offers a description of psychotic phenomena based on clinical experience and derived from prolonged therapeutic relationships. Moreover, it provides inferential ideas about unconscious functioning which are analysed with psychotic patients using interpretation over time. Consequently, it can contribute valuably to the understanding of personal vulnerabilities, the dynamic mechanisms an individual resorts to when facing unbearable affects or cognitions, and the supposedly disguised personal meaning or significance of psychotic phenomena (e.g. unsignalled metaphors in delusional beliefs).

Although the validity and efficacy of psychodynamic treatments for psychosis has received contrasting views (e.g. Hingley, 1997a; Mueser & Berenbaum, 1990), some of the psychodynamic concepts have been supported by cognitive research. For example,

the loss of the capacity to test reality has found parallels in the metacognition of reality testing in hallucinations (Bentall, 1990), whereas, the concept of projection has been substantiated by studies into the defensive function of persecutory delusions (e.g. Lyon et al., 1994). Moreover, there appears to be a promising trend towards the integration and further testing of valuable psychodynamic ideas such as defence mechanisms and the nature of cognitive distortions during information processing (e.g. Hingley, 1997b), a development within which the work reported in this thesis could also be suitably placed.

1. 4. Summary and integration.

In this Chapter, the main issues surrounding the relationship between trauma, dissociation and psychosis have been introduced and an outline of the aims of this thesis has been given. The methodology employed in the experimental investigation reported in Chapter 3 draws from paradigms used in cognitive psychology, which have been duly modified in order to address specific hypotheses. A review of the conceptualisation of psychosis and psychotic disorders has been presented, and the main clinical aspects and diagnostic classifications and limitations have been illustrated. Subsequently, the major psychological theories of psychosis have been reported with a description and critical evaluation of the key features for each of the approaches considered.

A common way of accommodating the disparity of findings and the multiplicity of factors proposed by exponents of differing theoretical orientations is that of presenting

some overarching, multifactorial model of psychosis, the most cited of which is probably Zubin and Spring's (1977) stress-vulnerability model. This and other similar models (e.g. Ciompi, 1988; Strauss & Carpenter, 1981) suggest that the development of a disorder is the result of the presence of a necessary but not sufficient predisposition (vulnerability or diathesis) for the disorder, and a similarly necessary but not sufficient stressor which interacts with the diathesis. Although stress-vulnerability models may appear very seducing in that they encompass all possible factors relevant to the aetiology and maintenance of psychotic disorders, they may be somewhat misguided for at least two reasons. Firstly, because of their overinclusive nature, they are able to incorporate and account for disparate manifestations of psychotic experiences under the unique umbrella of dubious diagnostic categories such as schizophrenia. Secondly, they seem to assume that the vulnerability factors are essentially biological and that the stressors are effectively environmental factors which increase the risk of symptoms emergence and modulate the course of the disorder. When psychological vulnerability is taken into account (e.g. cognitive deficits, cognitive distortions and misattributions), it seems to be done in the context of underpinning biological vulnerability (e.g. genetic, neurophysiological and/or neuroanatomical abnormalities), therefore giving the false impression that biological vulnerability takes precedence as a fundamental and necessary cause of psychosis.

An alternative way of adopting an integrative approach might be that of looking for points of contact amongst the different theoretical stances. This would involve the identification of conceptually related ideas and/or convergent evidence to be developed

into further hypotheses and tested in a systematic fashion. Such an approach would hopefully eliminate one further weakness of the stress-vulnerability models, that is, the provision of a comfort area within which investigators can pursue separate research programmes in isolation, with the danger of strengthening further the idea of (seemingly) unrelated, yet all important, vulnerability and stress factors.

As emerged from the review outlined above, one point of convergence appears to be the difficulty in reality testing identified by several investigators which affects people with psychosis; a disturbance that seems to be crucial in the development and maintenance of a range of psychotic symptoms, chiefly delusions and hallucinations (e.g. Bentall, 1990; Frith, 1992; Frosh, 1983; Hemsley, 1996; Morrison, 1998). This identified common ground provides us with the possibility of investigating the nature of some of the psychological processes involved in the disturbance of reality testing, namely, dissociation (as a hypothesised mechanism of defence from trauma and/or extreme negative affect) and the cognitive processes (i.e. attention and memory) on which it impinges.

As mentioned above, dissociation and its relationship with trauma and psychosis are central issues to this thesis and therefore they shall be introduced in the next Chapter.

Chapter 2

Trauma and dissociation.

"Come, blessed peace, we once again implore,
and let our pains be less, or power more."

(Alexander Brome, "The Riddle")

2. 1. Introduction.

In this Chapter we shall endeavour to articulate the two related concepts of psychological trauma and dissociative phenomena. The experience of potentially traumatic events in people's lives is quite common, with an estimated lifetime prevalence ranging from approximately 40 to 70% in the general population (e.g. Elliott, 1997; Kessler, Sonnega, Bromet, Hughes & Nelson, 1995) and, as mentioned previously in Chapter 1, up to 98% in individuals with psychosis (e.g. Mueser et al., 1998). Also dissociative experiences (e.g. absorption-imaginative involvement, depersonalisation, emotional numbing) appear to be common (approximately 80% in the general population), especially in traumatised populations (e.g. Kihlstrom, Glisky & Angiulo, 1994; Ray & Faith, 1995), to the extent that they have been deemed to be one of the primary features of traumatic stress reactions (e.g. Foa, Riggs & Gershuny, 1995; Herman, 1997). In this Chapter, we shall begin by introducing a broad evolutionary view

of trauma as a human stress response, in order to provide a theoretical context within which the psychological processes of *integration* vs. *sealing over* can be understood as basic adaptive vs. maladaptive reactions to the trauma of psychosis. Subsequently, we will present some of the current psychological theories of post-traumatic stress disorder (PTSD) in order to consider a number of possible psychopathological processes occurring following trauma. Finally, we shall progress to illustrate the nature and current understanding of dissociation, particularly as a response to traumatic stress, and will conclude by drawing attention to its relationship with trauma and psychosis.

2. 2. An integrative view of trauma.

In addressing the concept of trauma, one of the basic issues to be considered is its relationship with stress. A common sense model of their relationship is that trauma could simply be regarded as an extreme form of stress. However, this linear model is complicated by a number of factors. One of these is: how extreme must a stressful event be in order to constitute a traumatic experience? The DSM-III (American Psychiatric Association, 1980) officially recognised PTSD and distinguished trauma from stress by defining it as an event that is out of the normal range of usual human experience. However, this definition was deemed to be unsatisfactory for at least two reasons: *a*) as mentioned above, traumatic events are quite common in the general population; *b*) events are traumatic not because they are unusual and occur rarely, but rather because they overwhelm an individual's ordinary abilities to adapt to life. Consequently, this

conceptualisation of trauma was changed in DSM-IV (American Psychiatric Association, 1994) and a traumatic experience was defined as "exposure to an extreme traumatic stressor involving direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate" and to which an individual would respond with "intense fear, helplessness and horror" (p. 424). Although this definition recognises that a traumatic event need not be unusual and allows for an individual's subjective experience to be taken into account, it may also be considered too narrow in that it does not concede that threats to the self may be traumatic even if they do not involve bodily threats.

In line with this view, and in relation to the experience of psychosis, Jackson, Knott, Skeate and Birchwood (2004) have recently argued that the DSM-IV criteria for PTSD place too much emphasis on the facet of physical integrity at the expense of *psychological integrity*. Additionally, people with psychosis may also perceive themselves to be physically at risk due to paranoid delusions (e.g. persecutory), hallucinations (e.g. auditory, visual), coercive interventions (e.g. medication, seclusion), or from other psychotic individuals in a psychiatric ward. Therefore it is argued that both physical and psychological threats to one's integrity may constitute "exposure to an extreme traumatic stressor".

From an evolutionary framework point of view, stress is a state of arousal that occurs in response to environmental stressors and that leads to biopsychosocial changes which may bring about negative and/or positive consequences for an individual's adaptation and well-being (e.g. Christopher, 2004). In fact, stress appears to play a critical role not only in the emergence of bio and psychopathology, but also in emotional and cognitive development (e.g. Bjorklund & Pellegrini, 2002; McEwen, 2002). The adaptive or maladaptive nature of a particular stress response depends on a number of factors, but chiefly, on an individual's relationship with his/her environment. According to evolutionary psychology, most psychopathology derives from a dissonance or discontinuity between the *environment of evolutionary adaptiveness* (EEA; a statistical composite of the environment for which we are biologically best adapted) and the actual environment (Cosmides & Tooby, 1997). When an individual feels stress as a result of perceived threat or cognitive discontinuity, a stress response system is activated in an attempt to escape or eliminate the source of stress. This will bring about changes in the central nervous system that range from subtle modifications (e.g. necessary to learn a new task) to more profound alterations of neural networks associated with emotional and cognitive paradigm shifts (McFarlane, Yehuda & Clark, 2002). Therefore, a traumatic stress response is an evolutionarily inherited response to extreme states of arousal which would normally lead to some positive and adaptive effects called *post-traumatic growth* (PTG), rather than PTSD, in the form of a more integrated sense of self and philosophy of life, and closer relationships with others (e.g. Davis, Nolen-Hoeksema & Larson, 1998; Tedeschi, Park & Calhoun, 1996; 1998; Waysman, Schwarzwald & Solomon, 2001). This framework seems to be consistent with evidence that shows that only a

proportion of individuals (approximately 5-55%) exposed to a potentially traumatic event will go on to develop PTSD, depending on type of trauma and demographic group (e.g. Breslau, Chilcoat, Kessler & Davis, 1999; Ehlers, Mayou & Bryant, 1998; Kessler et al., 1995; Koenen, Stellman, Stellman & Sommer, 2003; Norris, 1992; Widom, 1999).

Nonetheless, in keeping with an evolutionary perspective, the hallmark symptoms of PTSD such as cognitive re-experiencing of a traumatic event (e.g. intrusive memories), arousal and hypervigilance, and protective behavioural/cognitive avoidance (including amnesia, emotional numbing and dissociation) can also be viewed as the basis of PTG and therefore as adaptive behaviours to extreme threats, which may become pathological under certain biopsychosocial conditions. In this respect, Eberly, Harkness and Engdahl (1991) have argued that hypervigilance towards the same or similar (via generalised anxiety to associated stimuli) events assists an individual to avoid the re-experiencing of a threat; emotional numbing and dissociation would allow an individual to distinguish between emotional and cognitive responses, thus allowing for more accurate and adaptive information processing; and cognitive re-experiencing enables an individual to learn from the traumatic event he/she experienced and to develop alternative more adaptive responses should similar events reoccur in future.

Related to this point, Christopher (2004) has hypothesised that the normal adaptive response to trauma can be conceptualised as *traumatic metalearning*, that is the process of shattering and reconstitution of metacognitive schema about the self, society and nature in which learning normally takes place. Consequently, PTG is seen as the forming

of new and more coherent cognitive schema, whereas, PTSD is seen as the result of a failure to modulate the normal adaptive trauma response with a more coherent and meaningful metaframework.

In this context, we shall define as traumatic an event that threatens one's physical and/or psychological integrity (including the experience of psychosis), that challenges an individual's fundamental beliefs about the self, others and/or the world, and that can not be dealt with by means of one's usual range of coping abilities. The extent to which an individual will perceive an event as being dissonant from his/her existing schema will determine the level of adjustment required in order to accommodate it within a reviewed more adaptive set of beliefs and coping skills. Such a stressor would normally result in an individual's experience of a cluster of trauma-related symptoms (e.g. dissociation, intrusive memories, hypervigilance, avoidance) and lead to either PTG and a more integrated sense of self, or PTSD (PP-PTSD, in our case) and sealing over.

The evolutionary framework is appealing in allowing us to set the concept of trauma of psychosis within a useful ecological context. Additionally, it is also valuable in highlighting the normally adaptive nature of human traumatic stress reactions, and in proposing common physiological, cognitive, behavioural and emotional processes of adaptive and maladaptive responses. However, this biopsychosocial view is largely grounded on studies of evolutionary biology geared towards the identification of determinants of phylogenetic adaptation to stress. As such, it does not provide an in depth understanding of some of the key psychological factors, such as risk and

protective factors that may influence whether an individual will develop PTSD rather than experience PTG, the mechanisms of maintenance of the disorder, and specific cognitive and emotional processes involved. Therefore, we shall now focus our attention on some of the most influential psychological theories of trauma and post-traumatic stress.

2. 3. Psychological theories of PTSD.

Given the high lifetime exposure to trauma and the usually devastating effects of experiencing psychotic symptoms acutely (during an episode) and residually (in between episodes), individuals with psychosis typically report a high degree of trauma-related symptomatology and may develop PP-PTSD (e.g. Jackson et al., 2004; Shaw et al., 2002).

Although several theoretical approaches exist that offer interesting insights into the nature of trauma and psychological traumatic stress reactions, in this Section we shall focus on cognitive theories of PTSD because they are probably the most fully developed and offer the greatest explanatory and predictive power (e.g. Dalgleish, 1999; 2004). As such, while not specifically developed to account for trauma-related symptoms in people with psychosis, a review of empirically based models of PTSD should provide us with a conception of key psychological processes implicated in the post-traumatic stress responses of our client group, as well as part of the theoretical basis on which to develop our hypotheses.

In the main, cognitive theories of PTSD tend to share the following propositions: *a*) individuals experience trauma with a set of pre-existing beliefs about themselves, the world and the future; *b*) the experience of trauma provides highly salient information that is incompatible with these pre-existing beliefs; *c*) unsuccessful attempts to assimilate this new information with pre-existing models of the self and the world may lead to pathological post-traumatic reactions (e.g. Brewin & Holmes, 2003); whereas, by extension, successful information processing which results in adaptive changes of existing models would produce PTG, as reviewed above. A similar view of trauma processing as part of psychosis has been proposed by Morrison et al. (2003).

This general conceptualisation of the emergence of PTSD is reflected in Horowitz's (1976; 1986; 1997) *stress response theory*. According to this theory, in response to a stressful or traumatic event and in recognition of loss, individuals will experience an initial emotional reaction in the form of crying out or shock. This is then followed by an attempt to assimilate the new trauma information with pre-existing knowledge. However, Horowitz argues that, at this point, most individuals will experience an information overload in which memories, thoughts and images of the traumatic event cannot be easily reconciled with prior beliefs. Consequently, two distinct processes are thought to come into play: one that tends to promote the resolving of the traumatic material by bringing it to mind, and a second one that mobilises psychological defence mechanisms that tend to avoid, suppress and pace the extent to which traumatic information comes into consciousness. Accordingly, individuals will oscillate between intrusion and avoidance of the trauma memory, and failure to process its information

and adjust previous models of the self, the world and the future will result in the development of PTSD.

Another account of the long term adjustment after the experience of trauma is provided by Janoff-Bulman's (1992) *theory of shattered assumptions*. According to this view, the most significant factors influencing an individual's response to trauma are certain basic assumptions about the self and the world. Specifically, these include a positive view of the self (e.g. worthy, competent, invulnerable), and a view of the world as benevolent, comprehensible, predictable and meaningful. Janoff-Bulman (1992) argued that PTSD is the result of these assumptions being shattered following the experience of a traumatic event and difficulties in updating them by cognitively reappraising and reframing the trauma.

Both these theories of PTSD could be thought of as cognitive-social frameworks, in that, they emphasise the likely wide impact of trauma in an individual's life, the readjustment he/she would need to make in order to integrate a traumatic experience into pre-existing views of the self and others, the role of his/her interpersonal context in facilitating or blocking this process, and the potential for PTG.

In contrast, information-processing theories focus on the (subjectively perceived as) traumatic event itself, how trauma-related information is represented, and how it is subsequently processed in the cognitive system. For instance, Foa, Steketee and Rothbaum (1989) have proposed a *fear network approach* to PTSD. The central idea is that traumatic events are represented in memory in the form of fear networks consisting

of interconnections between different nodes that contain information about the stimuli (both present and associated through conditioning), cognitive, emotional and physiological reactions, and behavioural responses to the events. Since one's basic assumptions of safety are typically overturned following trauma, causing the individual to become hypervigilant, the information contained within a fear network can be easily activated by triggering stimuli (e.g. reminders of the event) and reach consciousness (e.g. intrusive thoughts/images), which may lead the person to avoid and suppress the intrusions.

According to Foa and colleagues (1989), successful resolution of the trauma can only occur by weakening the overly strong associations between the nodes of the fear network and by integrating its information with the individual's existing memory structures. However, in order to achieve this, the network needs to be activated (e.g. via imaginal or in vivo exposure) and then modified by incorporating information that is incompatible with it.

This original network theory has been developed further by Foa and Riggs (1993) and Foa and Rothbaum (1998) in order to take into account beliefs present before, during and after the trauma, and also to include the role of appraisal in PTSD. Specifically, the authors argue that individuals with more rigid pre-trauma beliefs (either positive or negative) are more vulnerable to develop PTSD, in that, extremely positive beliefs would be contradicted and extremely negative beliefs would be confirmed by the experience of a traumatic event. Moreover, attention is drawn to the individual's negative appraisal of his/her responses during and after the trauma which might

exacerbate and reinforce negative beliefs, such as the self as incompetent and the world as dangerous.

The single level of representation of schema-based and associative network models of PTSD (e.g. Foa et al., 1989; Horowitz, 1997; Janoff-Bulman, 1992) has been criticised for being too simple and unable to capture the complexity of clinical (but also non-clinical) phenomena, such as emotionally laden vs. cold memories, or meaning that goes beyond what can be expressed verbally (e.g. Dalgleish, 2004; Power & Dalgleish, 1997; Teasdale & Barnard, 1993). In contrast, the *dual representation theory* of PTSD (Brewin et al., 1996) suggests that there are two distinct memory systems that operate in parallel and that store information in two different representational formats. The first type is called "verbally accessible memory" (VAM) and contains a narrative memory of (parts of) events that have received conscious processing. These are placed within a personal spatial-temporal autobiographical context and can be retrieved either automatically or using deliberate, strategic processes. The emotions that accompany VAM memories include "primary emotions" (experienced at the time of the event) and "secondary emotions" (generated following retrospective appraisal of the event).

The second type of memory system is called "situationally accessible memory" (SAM) and contains information obtained from a lower level perceptual processing of details of an event which have not received conscious attention (e.g. sounds, sights, smells, proprioceptive sensations). Since the SAM system does not use a verbal format, information on these memories cannot be easily communicated to others and is difficult

to control because we cannot regulate exposure to such stimuli. The emotions that accompany SAM memories are only those experienced during the event (i.e. "primary"). According to Brewin et al. (1996), PTSD is a hybrid disorder and recovery depends on the reduction of negative emotions present in the VAM system (i.e. resolution of negative beliefs) and the management of SAM memories (i.e. flashbacks). This last process would be achieved by re-encoding the SAM memories into the VAM format so that they can acquire a spatial-temporal context, can be placed in the past, and any reminders will trigger a retrieval competition which would end with the VAM system exerting control over the SAM system and the individual's emotional response.

Consequently, unlike what the other theories reviewed above assert, the dual representation theory proposes that traumatic memories do not need to be changed, instead, recovery is seen as the result of the introduction of retrieval competition between an old and a new, more controllable, memory of the trauma.

The latest major theoretical development concerning pathological reactions to trauma is Ehlers and Clark's (2000) *cognitive model* of PTSD. This model tends to pull together several of the concepts outlined above and is in many respects similar to the dual representation theory just reviewed. The authors maintain that PTSD arises when individuals perceive a sense of current threat due to the way they process traumatic information. This threat can be either an external threat to safety or an internal threat to the self and the person's future and it is the result of the individual's negative appraisal of the event, including its consequences, and of the very nature of the traumatic memory itself. More explicitly, Ehlers and Clark (2000) argue that pre-existing negative

experiences and beliefs increase the probability that individuals will respond to trauma in a way that highlights their inability to influence their fate, their vulnerability and helplessness, termed "mental defeat". Moreover, similarly to Brewin et al.'s (1996) proposition, it is suggested that trauma memories are poorly elaborated, lack spatial-temporal context, are processed perceptually rather than conceptually (cf. Roediger & McDermott, 1993), and are retrieved unintentionally. Finally, Ehlers and Clark (2000) propose that maladaptive behavioural strategies and cognitive processing styles are the core maintaining factors of PTSD.

It is apparent that there is a high degree of overlap among the cognitive theories of PTSD reviewed above (see Brewin & Holmes, 2003; Dalgleish, 2004; Dalgleish & Power, 2004 for in depth critical evaluations and recent developments). They underline the importance of several factors (e.g. personal beliefs, negative appraisal, altered memory functioning) that might render an individual more vulnerable to adopt maladaptive responses to traumatic events and progress along a pathological pathway to the development of PTSD. Later in this Chapter (Section 2. 5.) we shall see how these concepts may apply to psychosis, how they may be usefully employed to make predictions about the way individuals who have experienced one or more psychotic episodes may process trauma-related information, and how they may help account for different recovery styles adopted by individuals in the aftermath of a psychotic episode. However, beforehand we shall spend some time trying to articulate a view of dissociation, as this will constitute an important part of our investigation.

2. 4. Dissociation and its relationship to trauma.

The conceptualisation of dissociative phenomena has been the subject of much discussion and debate (e.g. Frankel, 1996; Kluft & Foote, 1999; Ross, 1996). Broadly, dissociation has been defined as a separation of mental processes (e.g. thoughts, perceptions, memories, emotions, identity) that are normally integrated and accessible to conscious awareness (Spiegel & Cardeña, 1991). This fragmentation or altered state of consciousness is thought to lead to a compartmentalisation of experiences and other psychological processes such as depersonalisation, derealisation, absorption, identity confusion, amnesia, disengagement, and emotional numbing. Consequently, dissociation has been conceptualised as a phenomenon that consists of a continuum of experiences present to some degree in most individuals (e.g. Ross, Joshi & Currie, 1990).

In everyday life, dissociation has been invoked to explain why an individual is able to conduct complex but routine activities (e.g. driving a car) whilst simultaneously engaging in other less predictable activities (e.g. holding a conversation). However, at the opposite end of the dimension, as found in clinical populations, dissociation can become a problem and contribute to psychopathology, in that, rather than simply involving the *automaticity* of over-learned behaviour, dissociation relates to the *unintentionality* of a process that leads to the inability to integrate the compartimentalised aspects of a given experience within consciousness, even though an individual may be willing to do so. Therefore, dissociation appears to disrupt two key elements of consciousness: *awareness* and *voluntary control* (Kihlstrom, 1984). Additionally, this conceptualisation of dissociation does not necessitate that the

separated components of an experience be entirely independent of each other. In fact, dissociated information or psychological processes, although unavailable to consciousness, can nonetheless have an interfering or facilitating (e.g. priming) effect on ongoing tasks.

An early model of dissociation that attempts to account for dissociative experiences is Braun's (1988) *BASK model*. This model proposes that an individual can separate, or dissociate, aspects (behaviour, affect, sensation, and knowledge) of a given experience so that he/she may not be aware of one or more of these, which would lead to dissociative experiences of varying degrees. Another attempt at explaining a wide range of dissociative phenomena is van der Hart, van der Kolk and Boon's (1996) *hierarchical model*. According to the authors, there are three levels of pathological dissociation: *a*) primary dissociation, which entails an individual's fragmented processing of traumatic events; *b*) secondary dissociation, which involves the individual perceiving a traumatic event without experiencing its full emotional impact; and *c*) tertiary dissociation which results in the development of separate identities. Both of these early models, however, appear to be rather descriptive in their nature and lacking explanatory power. As such, they are relatively tentative, do not propose specific mechanisms by which dissociative processes may operate, and do not lend themselves to the generation of specific predictions that may be empirically tested.

It has been pointed out that (pathological) dissociation appears to possess many of the attributes normally assigned to the defence mechanism of repression (Frankel, 1990).

However, other authors (Davies & Frawley, 1994; Singer & Sincoff, 1990) have tried to differentiate the two by defining repression as an active process through which the ego attains control over conflicting material which is pushed deep into the unconscious where it cannot be accessed; whereas, dissociation is defined as a process by which some aspects of an experience (typically traumatic) are "cordoned off" by severing their connection with the other components of the experience (e.g. separating thoughts and emotions), because they are too overwhelming to be processed and then repressed. Additionally, dissociation is seen as a fluctuating state of consciousness, therefore, dissociated material may also be only partially or alternately out of an individual's awareness.

This view of dissociation has led a number of authors to note parallels with hypnosis, which has been described as a state of controlled and structured dissociation (e.g. Butler, Duran, Jasiukaitis, Koopman & Spiegel, 1996; Putman, 1991; Spiegel & Cardeña, 1990; 1991). Indeed, Janet (1907) saw hypnosis and dissociation as being virtually synonymous and, more recently, Bliss (1984) has argued that the primary mechanism of dissociative identity disorder (DID) is an individual's spontaneous and unintentional misuse of self-hypnosis.

Pierre Janet was the first to study dissociation systematically and saw it as a psychological process with which individuals react to overwhelming trauma. He also argued that the memories of a traumatic event, although out of an individual's awareness, continued to affect his/her perception, mood and behaviour, and that in order to adapt to trauma he/she would need to assimilate the event to existing experiences,

whereas, continuing to dissociate would lead, over time, to the emergence of psychopathology. In recent years, Janet's ideas on trauma and dissociation have been re-evaluated and integrated into contemporary models of dissociation and trauma (cf. also Section 2. 3., above). For instance, Putnam (1993) has proposed a number of protective functions of dissociation: *a*) automatisation of certain behaviours; *b*) resolution of irreconcilable conflicts; *c*) escape from the constraints of reality; *d*) isolation of catastrophic experiences; *e*) cathartic discharge of certain feelings; *f*) analgesia; and *g*) alteration of the sense of self, so that a traumatic event is experienced as if it was not really happening to oneself.

Similar views have been proposed, from a psychodynamic perspective, that articulate dissociative processes as reflecting intra-psychic defences, such as denial (e.g. pretending that repeated trauma is not occurring) and splitting between a traumatised and a coping-self, in order to diminish the impact of trauma (e.g. Fonagy, 1991; Mollon, 1996). However, as a defence, dissociation may be seen as having a double edge: it may be adaptive in the short-term by providing protection from the immediate impact of traumatic experiences, but may result in the long-term fragmentation of the self, which may lead to further anxiety.

Other authors (e.g. Bentovim, 2002; Kluft, 1996; McIntee & Crompton, 1997; Putman, 1997) have used a developmental perspective to formulate the traumatic origins of dissociative processes within disorders such as DID and borderline personality disorder (BPD), and have suggested that childhood trauma (e.g. sexual, physical, emotional abuse, neglect) leads to dissociation and failure to achieve the major developmental task

of integrating discrete states of consciousness, as well as impacting on fundamental functions, such as development of attachments, emotion regulation, and development of an adequate sense of self. Indeed, there is evidence to suggest that childhood trauma is predictive of pathological dissociation in adult clinical and non-clinical samples (e.g. Anderson, Yasenik & Ross, 1993; Brunner, Parzer, Schuld & Resch, 2000; Carlson, Armstrong, Loewenstein & Roth, 1998; Irwin, 1999; Kroll, Fiszdon & Crosby, 1996; Lipschitz, Kaplan, Sorkenn, Chorney & Asnis, 1996; van der Kolk et al., 1996).

Nonetheless, the presence of a simple linear relationship between trauma, dissociation and trauma-related psychopathology has been questioned by some authors (e.g. Frankel, 1996; Gershuny & Thayer, 1999; Merckelbach & Muris, 2001; Merckelbach, Horselenberg & Schmidt, 2002; Tillman, Nash & Lerner, 1994). The main arguments put forward are that other factors may intervene in the emergence of psychopathology, and that the link between trauma and dissociation may be mediated by other variables, such as fear of death and fear of loss/lack of control. However, while the first argument appears to be valid (as supported also by the literature reviewed in Section 2. 3.), to date, the latter is based on theoretical speculations or studies that looked at the mediating factors of fantasy proneness and absent-mindedness in the relationship between retrospective self-reported trauma and dissociation in student samples, rather than clinical populations. Even so, structural equation modeling provided equivalent goodness-of-fit statistics for both the mediated and the more direct linear models (Merckelbach et al., 2002).

Summarising several strands of research, Butler, Duran et al. (1996) proposed an understanding of the development of dissociative symptomatology based on a *diathesis-stress model.* According to this model of dissociation, the diathesis is the capacity of an individual to dissociate (or hypnotisability), which has been shown to be normally distributed in clinical and non-clinical samples, with relatively higher tendencies for females, an increase from mid-childhood to adolescence, and subsequent decline with age (e.g. Bernstein & Putnam, 1986; Ross, et al., 1990; Ross, Ryan, Anderson, Ross & Hardy, 1989; Torem, Hermanowsky & Curdue, 1992). Moreover, in support of this diathesis, recent studies have also found a significant positive correlation between dissociative tendencies and the personality trait of neuroticism in both clinical and non-clinical populations (Goldberg, 1999; Groth-Marnat & Jeffs, 2002), as well as state and trait anxiety (Wolfradt & Meyer, 1998), and schizotypy (Bauer & Power, 1995). On the other hand, what constitutes the stress component in this model is a traumatic experience due to either an external event or intra-psychic distress. The authors argue that severe traumatic experiences during childhood development are particularly likely to increase dissociative processes. Especially in the face of repeated trauma, these defensive processes may become too important to fade and decline during late adolescence, leaving an individual entrenched in a hypertrophied defence style, which may generalise and be used indiscriminately, to include also non-abusive or non-traumatic situations. This "auto-hypnotic process" is thought to result in pathological dissociation and cause disruption in the domains of perception, behaviour, will, affect, memory, and identity (i.e. interfere with the content and control of awareness).

Recently, a *cognitive model* has been put forward in an attempt to elucidate the processes implicated in dissociative phenomena, based on Beck's (1996) cognitive theory of personality and psychopathology (Kennedy, Clarke, Stopa, Bell, Rouse, Ainsworth, Fearon & Waller, 2004). Beck (1996) argued that personality is a composite of *modes*, defined as a set of schemas in charge of processing cognitive, behavioural, affective, and physiological information, as well as generating responses. Moreover, he proposed that *orienting schemas* automatically (i.e. without conscious effort) process internal and external events and activate different modes accordingly, so that in normal circumstances there is a smooth exchange of information between schemas and switching between personality modes depending on the context, as appropriate.

Kennedy et al. (2004) suggest that dissociation is the result of inhibitory "decoupling" of mental processes occurring at three different stages. At stage I, we can find *automatic dissociation*, which is the result of preconscious decoupling of the links occurring at the orienting schema level between perception, emotions and cognitions. This is a mechanism that inhibits the early associative stage of information processing, when material can be identified as being threatening, and may result in fragmented memory traces of an event. Dissociation at this level may lead to de-contextualised activation of fragments which would be manifested, for example, as visual or auditory hallucinatory experiences. The authors draw parallels between this mechanism and van der Hart et al.'s (1996) primary dissociation, as well as Brewin et al.'s (1996) SAM system.

At stage II, we can notice *within-mode dissociation*, which involves the strategic decoupling of the associative links between schemas (cognitive, behavioural, affective, and physiological) within a given mode. Dissociation at this level has implications for

storage and retrieval of information and may result in cognitive symptoms (e.g. intrusive thoughts, mind going blank), behavioural symptoms (e.g. ritualistic behaviour, re-enactment), affective symptoms (e.g. emotional flattening), and physiological symptoms (e.g. pain, analgesia, loss of function). The authors compare the cognitive element of this mechanism to Brewin et al.'s (1996) VAM system, and the affective element to van der Hart et al.'s (1996) secondary dissociation. Finally, at stage III we can observe *between-mode dissociation*, which is the result of the partial or total decoupling of different modes. This type of dissociation can take place to varying degrees, ranging from minor decoupling resulting in state-switches characteristic of BPD, depersonalisation and derealisation, to more extreme forms resulting in psychogenic amnesia and multiple or alter personalities characteristic of DID. As such, it is comparable to van der Hart et al.'s (1996) tertiary dissociation.

This new cognitive model of dissociation has not yet been validated by much supporting evidence. In fact, it is based on previous models which are not entirely corroborated and appears to suggest a quasi-linear progression from one stage of dissociation to the next, without stating clearly what the relationship between the three different types of dissociation is. However, it appears to have more explanatory power in comparison with previous models, and it makes some explicit hypothetical predictions based on previous research, which may be empirically tested. Consequently, in our investigation we shall draw more heavily on it compared to previous models of dissociation.

The literature reviewed in this Section brings to light a number of important points: *a*) the broad presence of dissociation from non-pathological to pathological levels in non-clinical and clinical populations; *b*) the intricate relationship between traumatic and dissociative experiences; *c*) the plausible defensive function of dissociation in attenuating trauma-related distress; *d*) the disruptive effects of dissociation on cognitive processes; and *e*) the potentially important role played by dissociative processes in the exacerbation and/or maintenance of trauma-related symptomatology as a result of their prolonged unintentional "misuse".

Accordingly, and for the purpose of this study, we shall define dissociation as a multidimensional phenomenon that leads to the fragmentation of (typically) trauma-related information processing, the compartmentalisation of experience at different levels (e.g. Kennedy et al., 2004), and as having the main defensive function of lessening/avoiding an individual's psychological distress in relation to the same traumatic events. As such, pathological dissociation is hypothesised to occur predominantly in individuals with a history of trauma, especially during the experience of potentially threatening events. Over time, it becomes reinforced and develops into a maladaptive way of coping, which results in the disruption of awareness, voluntary control, and contributes to the maintenance of trauma-related symptoms.

In the next Section we shall attempt to integrate the theoretical models and empirical evidence reviewed thus far (with particular emphasis on Brewin et al.'s (1996) and Kennedy et al.'s (2004) cognitive models of PTSD and dissociation), and to evaluate their relevance to psychosis.

2. 5. The link between trauma, dissociation and psychosis.

As we have seen above, the occurrence of traumatic life events and dissociative experiences in the general population is relatively common, and a strong link between the two has been proposed (e.g. Gershuny & Thayer, 1999). We have also drawn attention to the fact that psychotic symptoms (e.g. hallucinations and delusions) are also experienced in the general population (e.g. Kendler et al., 1996), especially in stressful or traumatic circumstances (e.g. following bereavement, in extreme sensory deprivation, in hostage situations). Moreover, we have reported evidence suggesting that dissociation and schizotypy exist as personality traits in the general population and that they are positively correlated (Bauer & Power, 1995; Myin-Germeys, Krabbendam & van Os, 2003; Pope & Kwapil, 2000; Startup, 1999; Waller, Putnam & Carlson, 1996). Therefore, it would seem reasonable to hypothesise that the experience of perceived stressful or traumatic life events is bound to increase the likelihood of the co-occurrence of both dissociative and psychotic experiences in the same individual.

In recent years, several authors have observed a high prevalence of PTSD symptomatology in people with psychosis (e.g. Frame & Morrison, 2001; McGorry et al., 1991; Meyer et al., 1999; Mueser et al., 1998; Mueser, Salyers, Rosenberg, Ford, Fox & Carty, 2001; Neria, Bromet, Sievers, Lavelle & Fochtmann, 2002; Resnick et al., 2003; Seedat et al., 2003). Specifically, in the clinical samples examined, the prevalence of PTSD ranged from 11 to 67%, and PTSD symptoms were related to three main factors: *a*) the presence of past trauma (especially in childhood); *b*) the experience of

psychosis; and *c*) hospitalisation. Moreover, levels of distress and intrusive memories of such negative events have been found to be significantly related to high levels of anxiety and dissociative symptoms in this client group (Shaw et al., 2002).

Whilst the frequent presence of post-psychotic PTSD, as well as of post-psychotic depression (e.g. Rooke & Birchwood, 1998), seriously questions the adequacy of the concept of "non-affective psychosis" (cf. Birchwood, 2003), it has been noted that the way in which individuals deal with the trauma of psychosis, although idiosyncratic, tends to vary within a limited number of roughly distinct patterns (Fowler et al., 1995). Many people who experience a psychotic episode react by resigning to the "fact" that their lives have changed forever and by assuming the social role of a chronic mental patient. They may become engulfed in the sick role, be overly dependent, and present with a range of emotional reactions (e.g. anxiety, depression, shame, anger, helplessness, hopelessness, entrapment, suicidal ideation), which may be indicative of what Ehlers and Clark (2000) termed "mental defeat" in relation to severe reactions to trauma.

Other people cope with the trauma of psychosis by denying the presence of any difficulties or *showing* no awareness. This way of coping has similarities with the recovery style referred to as *sealing over* by McGlashan (1987): a process by which an individual views his/her psychotic experiences as alien, avoids any attempts at investigating/understanding his/her symptoms, minimises them, and isolates them by conscious suppression or unconscious repression. It is argued here that this psychological process may be understood as a sustained state of disbelief comparable to

Horowitz's (1997) description of the initial shock response to trauma (see also Jackson, Tait, Birchwood & Trower, 2002). Moreover, we propose that this coping mechanism may be mediated by a state of dissociation that ranges from within-mode (or secondary) to between-mode (or tertiary) depending on the degree of sealing over. Specifically, within-mode dissociation would decouple the cognitive and affective components of a traumatic event which, as argued by Eberly et al. (1991) from an evolutionary perspective, has the adaptive value of allowing an individual to disentangle the emotional from the rational aspects of the event and foster self preservation. Between-mode dissociation, on the other hand, would have the adaptive function of preserving the "healthy self" of an individual. Putnam (1993) has argued that among the defensive functions of dissociation is the alteration of the sense of self, so that a traumatic event is experienced as if it is not really happening to oneself, and a sense of escape from the constraints of reality. Given the trauma of psychosis, it is proposed that individuals may well attempt to protect their sense of self by dissociating. However, symptoms of depersonalisation and derealisation would necessarily encumber one's relationship with oneself and the world, seriously compromising an individual's ability to test reality.

Another way of dealing with the emergence of a psychotic episode is accepting one's predicament and being able to adapt and make lifestyle adjustments in order to maximise independent functioning. This coping pattern is similar to McGlashan's (1987) notion of *integration*, characterised by an individual's awareness of his/her mental state before, during and after a psychotic episode, curiosity about his/her experience, and commitment to understanding with the help of others. In this case, trauma-related

symptoms appear to drive some individuals towards the generation of meaning of their psychotic episode and they are likely to experience PTG. This recovery style has been associated with better engagement with mental health services and better outcome (e.g. Tait, Birchwood & Trower, 2003; 2004; Thompson, McGorry & Harrigan, 2003). It is argued that individuals who use integration as a coping style may make less use of dissociative processes, at least at the between-mode/tertiary level.

Although McGlashan (1987) maintained that sealing over and integration are best conceived as enduring personality traits, other authors have pointed out that an individual's pattern of reactions to psychosis is variable over time, as it is likely to fluctuate between resignation, denial and acceptance (Fowler et al., 1995). This is supported by Thompson et al.'s (2003) study of outcome in first-episode psychosis, who found that recovery style changed over time; and by Lindbom-Jakobson and Lindgren's (2001) study of people who had experienced political persecution and torture, who observed that both coping styles were parts of the working through of traumatic events. In particular the authors found that it was easier for individuals to integrate hate and aggression evoked by the torture trauma, than feelings of guilt and shame, which were dealt with preferentially by sealing over. These findings are consistent with the fact that individuals with psychosis who seal over tend to exhibit higher levels of depression, make more negative self-evaluations, report negative early experiences (i.e. uncaring parents) and generally tend to appraise greater loss and shame in their psychosis, compared to those who integrate their psychotic experiences into their wider life experiences (Birchwood, Iqbal, Chadwick & Trower 2000; Drayton, Birchwood &

Trower, 1998; Iqbal, Birchwood, Chadwick & Trower, 2000). To this effect, Birchwood and colleagues have argued that "sealing over is an adaptive but ineffective strategy for coping with the trauma of psychosis".

This analysis is also concordant with the psychodynamic view of psychosis, which emphasises the importance of traumatic childhood experiences in the origins of the disorder, and the overuse and reliance on immature defence mechanisms to protect the fragile self from anxiety (i.e. unbearable affect and cognition); and indeed, dissociation has been seen as part of an immature defence style (Butler, Duran, et al., 1996).

Relating once again these types of recovery styles to conceptualisations of trauma, it appears that the distinction between integration and sealing over is analogous to Horowitz's (1997) proposition that two psychological processes are in action following trauma: one that tends to promote the resolving of the trauma by bringing it to mind, and a second one that mobilises defence mechanisms that tend to avoid it. It seems therefore reasonable to suggest that individuals will differ in their response to the trauma of psychosis by integrating their experience or by sealing over and avoiding it depending on their current capacity for adaptation, which will rest on a number of protective and risk factors (e.g. social support, additional life stresses, pre-morbid coping style, etc.).

Connected to this point and referring to defence mechanisms, Vaillant (1994) argued that often psychopathology is not merely the result of life stresses, but also of an individual's idiosyncratic response to his/her stressful environment. Consequently, a clinician who includes the client's defensive style as part of the formulation will be in a

better position to understand what may initially appear most irrational about the client, and to recognise the adaptive and maladaptive value of the client's defensive distortions of his/her inner and outer reality.

Given the intricate relationship between trauma and psychosis, Morrison et al. (2003) have recently reviewed current research literature on the topic, and have suggested that there is sufficient evidence to support, at least in part, three alternative views of their link. The first of these views is that experiencing a psychotic episode, especially for the first time, is a traumatic life event and, as such, it may cause PTSD in some individuals. Clinical case studies describe the terrifying nature of psychotic episodes (e.g. Shaner & Eth, 1989). The subjective experience of "going mad" or "losing one's mind", the highly distressing nature of hallucinations and delusions, and the experience of psychiatric services (e.g. compulsory hospitalisation, being held in a locked ward with other acutely psychotic people, being forcibly medicated), clearly represent major traumatic life events for which post-trauma conceptualisations would apply, whether these are experienced for the first time or constitute a relapse. In fact, the psychological implications for an individual who develops psychosis, at a personal level and within his/her interpersonal context, are the same as the ones described in various theories of PTSD reported above. For instance, Janoff-Bulman's (1992) notion of shattered pre-existing assumptions about the self, the world and the future, would very much apply to people developing psychosis at any point in their lives, and especially so at a young age.

The second view proposed by Morrison et al. (2003) is that trauma may cause psychosis in some individuals. This idea is based on the observation of high prevalence rates of trauma in people with psychosis (reported above), and the influence of negative life events in precipitating psychotic symptoms, although social and cognitive variables may modulate their relationship (e.g. Bebbington, Wilkins, Sham, Jones, van Os, Murray, Toone & Lewis, 1996; Bebbington, Wilkins, Jones, Foerster, Murray, Toone & Lewis, 1993; Myin-Germeys, Krabbendam, Delespaul & van Os, 2003; Norman & Malla, 1993). Moreover, it has been suggested that the negative subjective experience of prodromal and residual symptomatology in itself may also act as cumulative traumatic stress, which may aggravate symptoms and lead to a full-blown psychotic episode (Stampfer, 1990).

We argue that, following the resolution of an acute psychotic episode, the experience of delusions and/or hallucinations may be considered a prime source of cumulative traumatic stress. The individual may have to deal with this internally generated traumatic information typically laden with negative affect on a daily basis for protracted periods of time. In this sense, the experience of psychosis may be thought of as the *internalisation of traumatic events*, and the establishment of a quasi-perpetual self-generating cycle of trauma. Accordingly, Ehlers and Clark's (2000) model of PTSD may be invoked to account for PTSD symptoms in individuals with psychosis arisen as the result of their perception of external or *internal* threat to the self and their future.

The third possibility entertained by Morrison and colleagues (2003) is that PTSD and psychosis may be comparable disorders and part of a spectrum of responses to traumatic life events. This hypothesis is partly based on observations regarding the similarity of symptoms of PTSD and psychosis. Specifically, symptoms such as increased levels of arousal, hypervigilance, interrupted sleep, emotional numbing, detachment from others, derealisation, and general neglect, appear to be common to the two disorders; whereas, intrusive thoughts, images, and flashbacks, are often experienced in the form of auditory, visual, olfactory, or tactile hallucinations and are accompanied by paranoia (e.g. Butler et al., 1996).

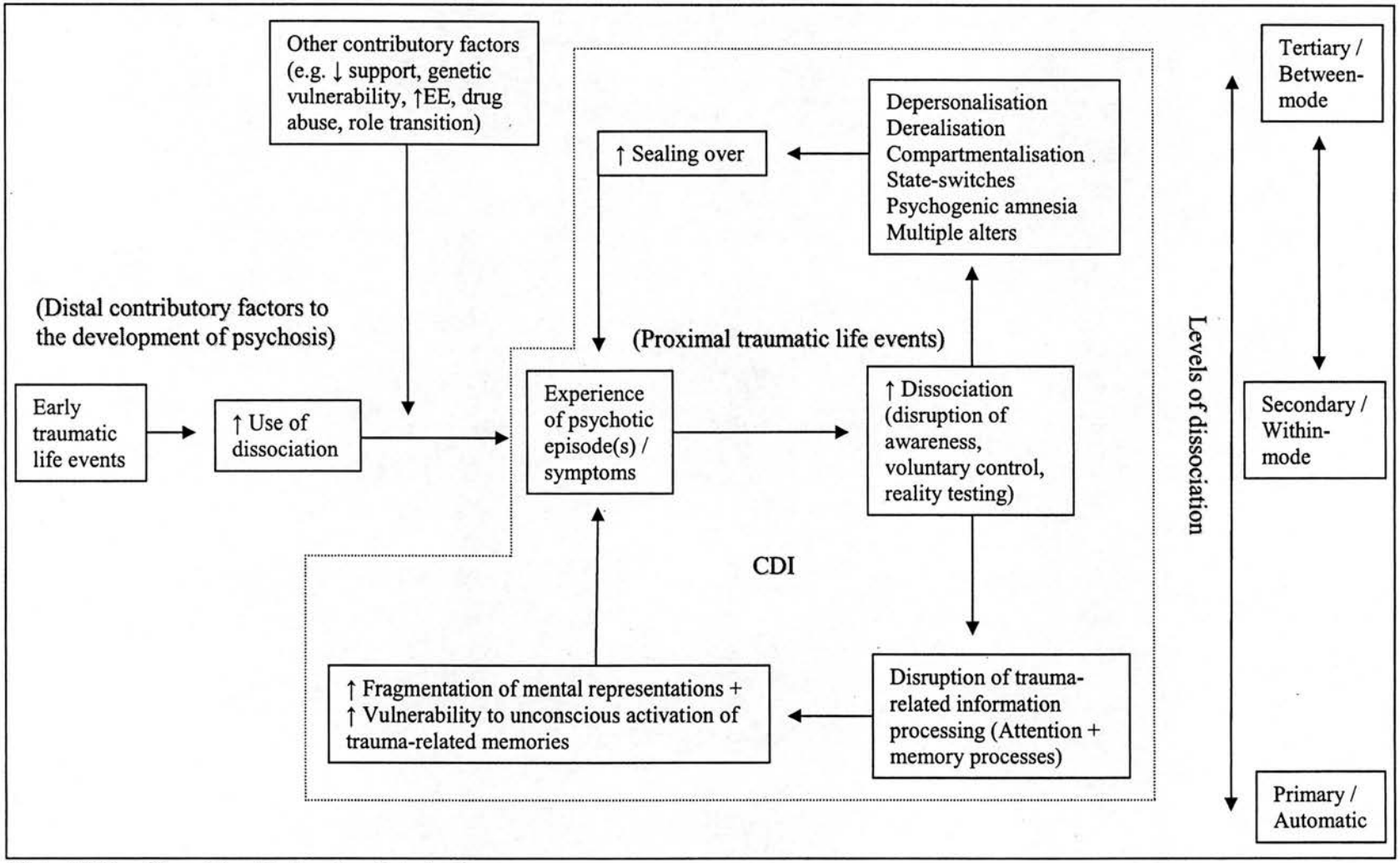
Both Brewin et al. (1996) and Ehlers and Clark (2000) propose that trauma memories, having received no or little conscious attention, are poorly elaborated, lack spatial-temporal context, and are retrieved unintentionally. This seems to be consistent with the view that dissociated information is patchy, lacks the normal contextual links and may come to consciousness involuntarily. Moreover, given these attributes, trauma-related information may appear to come out from nowhere and feel alien; experiences that individuals with hallucinations are well acquainted with.

Furthermore, Morrison et al. (2003) point out that both disorders are positively related to levels of dissociation (typically measured by self-report questionnaires such as the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986)), and include common cognitive and behavioural maintaining processes, such as negative appraisal of intrusions, negative attributional biases, a "search for meaning", selective attention, avoidance and other safety behaviours.

Despite the inherent methodological difficulty in differentiating overlapping concepts and the risk of a circular argument which culminates with the construction of a conclusive "integrative" model (cf. Morrison et al., 2003), the main tenet of this thesis is that dissociation plays a pivotal role in the relationship between trauma and psychosis, particularly in relation to the formation and maintenance of symptomatology. Specifically, we hypothesise that the psychological processes by which this developing and exacerbating/maintaining mechanism may operate rely heavily on the cognitive by-products of the defence mechanism of dissociation, and that Brewin et al.'s (1996) dual representation theory of PTSD and Kennedy et al.'s (2004) cognitive model of dissociation provide an account of some of the specific cognitive underpinnings of these processes.

Figure 2. 1., below, gives a graphical representation of the proposed model of trauma, dissociation and psychosis. In the course of Chapters 1 and 2, we have reported evidence which suggests that early traumatic life events, as experienced by the great majority of individuals with psychosis, may render individuals susceptible to the use of immature defence mechanisms (dissociation included) when dealing with difficulties (e.g. Butler, Duran, et al., 1996; Frosh, 1983). This distal vulnerability, together with other contributory factors identified by several lines of investigations, is thought to lead some individuals to the development of psychosis (e.g. Bentall, 2003). We have also seen that psychotic symptoms, such as hallucinations and delusions (resulting from projection or misattribution) may in themselves serve the defensive function of maintaining a positive view of oneself (e.g. Lyon et al., 1994; Morrison et al., 1995).

Figure 2. 1. A cognitive model of trauma, dissociation and psychosis.



Note. CDI = Cognitive-Defensive Interlock.

It has also been argued that the experience of psychosis constitutes a traumatic life event in its own right and that, as such, it may lead to the development of trauma-related symptomatology, including dissociative processes (e.g. Jackson et al., 2004; Morrison et al., 2003; Shaw et al., 2002). On the other hand, although dissociation occurring at different levels of information processing has been identified as a psychological defence against trauma-related distress, it also has the effect of interfering with an individual's content and control of awareness, and his/her ability to test reality (e.g. Butler, Duran et al., 1996; Gershuny & Thayer, 1999; Kennedy et al., 2004; van der Kolk et al., 1996), which has been hypothesised to be crucial in the development and maintenance of psychotic symptoms (e.g. Allen et al., 1997).

We propose that dissociative processes in response to trauma are instrumental in the formation and maintenance of hallucinations and delusions, and that two (not mutually exclusive) different routes, determined by the level at which they operate, can be identified. The first of these routes (bottom part of Figure 2. 1.) is hypothesised to be mediated by dissociation occurring at a primary/automatic level, and that it is part of a primitive subcortical evolutionary inherited traumatic stress response (cf. Sloman, 1996). At this level, (peri-traumatic) dissociative processes act at an early stage of information processing by severing associative links between perception, cognitions and emotions, and by fragmenting the mental representation of traumatic experiences (e.g. Brewin et al., 1996; Ehlers and Clark, 2000). The disruption of normal attentional and memory processes, the resulting disintegrated representational format of events, and the reduced awareness and voluntary control (all "side effects" of the defensive function of

dissociation) increase an individual's vulnerability to the unconscious de-contextualised activation of trauma-related memories, which are experienced as further hallucinations and delusions (cf. Butler et al., 1996). In turn, the subjective negative appraisal of the psychotic symptoms and the generation of further traumatic distress ("side effects" of the defensive functions of hallucinations and delusions) are expected to kindle further dissociation.

The second route (top part of Figure 2. 1.) is hypothesised to hinge on within-mode/secondary and between-mode/tertiary levels of dissociation (Kennedy et al., 2004; van der Hart et al., 1996) and to rely on a more developed cortical response system that involves reasoning (cf. Sloman, 1996). In this case, dissociative processes operate by decoupling the associative links between schemas (cognitive, behavioural, affective, and physiological) and by compartmentalising different personality modes, to the extent that an individual is able to either attenuate or deny the impact of the experience of psychosis in his/her life. However, this contributes to a varying degree of sealing over adopted by an individual, which is a recovery style that has been associated with lower personal resilience, poorer engagement with mental health services and higher risk of relapse (Tait et al., 2003; 2004).

In summary, it is hypothesised that the adaptive and maladaptive defensive functions of dissociation on the one hand, and of delusions and hallucinations on the other, are likely to combine and develop an entrapping psychological mechanism for individuals with psychosis. We shall refer to this mechanism as the *cognitive-defensive interlock* (CDI): a

dynamic feedback loop that relies on the cognitive by-products of evolutionary developed defence mechanisms (cf. Teasdale & Barnard's (1993) *depressive interlock* within the interacting cognitive subsystems (ICS) model, and Gumley, White & Power's (1999) adaptation of this model to psychotic disorders). It is proposed here that the CDI is a major contributor to the maintenance of psychotic symptoms, in that, the interaction of dissociative, hallucinatory and delusional processes establishes a self-perpetuating cycle that acts to maintain a persistent negative set of affective states (e.g. anxiety, depression, shame, anger). Of course, all of these hypothesised processes need empirical verification.

2. 6. Summary.

As intended, we have started this Chapter by giving a broad overview of trauma within which we have placed existing influential cognitive theories of PTSD. We have then reviewed current models of dissociation and have related them to the view of trauma and traumatic stress reactions outlined above. Finally, we have provided an evaluative synthesis of the theoretical approaches and ideas considered so far and have extrapolated convergent conceptualisations of trauma, dissociation, and psychosis in order to disentangle some of the plausible processes underlying their relationship. In doing so, we have also put forward a number of theoretical proposals, some of which will constitute the basis for more specific hypotheses to be tested experimentally in our investigation reported in the next Chapter.

Chapter 3

An experimental investigation of trauma, dissociation and psychosis.

"When vain desire at last and vain regret
go hand in hand to death, and all is vain,
what shall assuage the unforgotten pain
and teach the unforgetful to forget?"

(Dante Gabriel Rossetti, "The House of Life")

3. 1. Introduction.

In the course of the previous two Chapters, we have reviewed current literature on psychosis, trauma and dissociation and have endeavoured to build the case for the crucial role played by dissociative processes in the contribution towards the development and maintenance of psychotic symptoms (chiefly, hallucinations and delusions) in response to the experience of traumatic events.

From our review of cognitive models of PTSD and dissociation, we have also seen that the format of the mental representations of trauma-related information is a key feature of the postulated mechanisms of symptom formation and maintenance. Therefore, from an information processing point of view, the basic cognitive processes underlying any

mental activity, namely, attention and memory, are of primary relevance for our understanding of these phenomena.

In this Chapter, we report an experimental study of attention and memory for trauma-related, positive, and neutral information in a sample of people with psychosis compared to a matched healthy control group. We also used measures of current psychological impact of trauma, dissociation, symptomatology and recovery style in our experimental group in order to examine their relationship, and also their potential impact on the participants' performance on the experimental tasks. With these two aims in mind, we attempt to present some initial empirical testing of the two routes outlined earlier in our CDI model by which dissociative processes are hypothesised to contribute to symptom formation and maintenance. However, before the presentation of the empirical work we shall review some of the relevant literature in the field of attention, memory and dissociation.

3. 2. Review of relevant experimental research.

One of the tenets of cognitive psychopathological theories is that differences in how individuals process information (emotional information, in particular) may play a crucial role in the aetiology, development, maintenance, and treatment of emotional disorders (e.g. Mathews & MacLeod, 1994). Consequently, over the past two decades researchers have increasingly turned to information processing paradigms derived from

experimental cognitive psychology in order to understand attentional and mnemonic biases occurring at different levels of information processing.

One of the tasks commonly employed in order to investigate attentional processes is an emotional adaptation of the classic Stroop colour-naming task (Stroop, 1938) known as the *emotional Stroop task.* In this task, participants are typically shown coloured words that are emotionally laden or neutral and are asked to name the colour (or press a corresponding coloured key) of each word while attempting to ignore the word itself. Several authors have applied this paradigm with a number of different client groups (meeting diagnostic criteria for particular anxiety disorders), and have found that participants are generally slower at naming the colour of words associated with their personal concerns (compared to neutral words), showing selective attention for emotionally relevant stimuli (for a review, see Williams, Mathews & MacLeod, 1996). This is the case also for individuals with PTSD who appear to show a particularly large interference effect (e.g. Buckley, Blanchard & Neill, 2000; McNally, 1995; Williams et al., 1996).

To the best of our knowledge, the only study that has explicitly examined the effect of dissociation on attentional processes is DePrince and Freyd's (1999) investigation in two groups of college students ($N = 54$ in each group) selected on the basis of their high or low scores on the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986). The authors assessed attention and memory performance in two conditions. The first consisted of a standard emotional Stroop task (selective attention) followed by an

incidental free recall task; whereas, the second condition consisted of a divided attention emotional Stroop task, in which participants were asked to name the colour of each word as quickly and accurately as possible, while also remembering the words for a memory test at the end of the task, which was in fact followed by a free recall task. Results indicated that the high-DES group exhibited more interference on the selective attention version and less interference on the divided attention version of the emotional Stroop task, and also recalled fewer emotionally laden words (especially in the second condition) compared to the low-DES group. The authors interpreted these findings by suggesting that high-dissociators may perform better when dual-tasking than low-dissociators, who perform best when focusing their attention. Given the relationship between trauma and dissociation, and adopting an evolutionary view, DePrince and Freyd (1999) proposed further that individuals who experience traumatic life events and dissociate, may learn to dual-task as a way of managing and controlling the flow of incoming information, that is, keep information that is at odds with survival goals away from consciousness. However, their study was carried out with college students rather than a clinical sample, and it did not include any measure of trauma.

In contrast, a number of studies have investigated memory processes in clients with a diagnosis of PTSD or DID, which have also included a measure of dissociation.

Research on memory and PTSD seems to indicate the presence of implicit and explicit memory biases favouring trauma-related material, but also confusion, disorganisation, and forgetting of specific autobiographical memories (e.g. Buckley et al., 2000; McNally, 1995). These seemingly contrasting findings could be interpreted as reflecting

the alternation of arousal and avoidance states, or the two psychological processes proposed by Horowitz (1997), or the relative influence of the SAM and VAM systems (Brewin et al., 1996), or a combination of the three.

Because of this pattern of results, recent investigations have used the experimental task known as *directed forgetting task* (DFT), to assess the ability of people with PTSD to engage in selective forgetting (or remembering) of stimuli (usually lists of emotionally laden and neutral words) that participants have been instructed to remember or to forget (e.g. Johnson, 1994). Typically, there are two versions of this task that vary in the way the stimuli to-be-remembered (TBR) or to-be-forgotten (TBF) are designated. In the list method, halfway through the task, participants are instructed to forget the first half of the list of words and to remember the ones presented during the second part of the task. In the word method, participants are instructed to remember or to forget each word immediately after individual presentations. Using either method, at the end of the task, participants are given a surprise recall test for the entire set of stimuli (i.e. TBR and TBF). Directed forgetting occurs when participants recall less of the TBF than of the TBR set of words. It has been suggested that the directed forgetting effect (DFE) is likely to be due to retrieval inhibition when the list method is used, since a reasonable amount of processing would have already been allocated to the TBF words, once the instructions to forget are given. On the other hand, when the word method is used, the DFE is likely to be due to differences in encoding/rehearsal and storage, since the cue to remember would direct participants to continue to process a stimulus, whereas, a cue to forget would direct participants to discontinue the processing (Basden, Basden &

Gargano, 1993; MacLeod, 1999). However, whichever processes might be responsible, a DFE is usually obtained only when explicit but not implicit memory is tested. *Explicit memory* refers to conscious recollection of previously presented information. Conversely, *implicit memory* refers to facilitation in task performance that is attributable to information acquired during a previous study phase although no conscious reference to this information is made (e.g. Schacter, 1987). Consequently, these two types of memory are also referred to as *conscious* and *unconscious*, respectively.

Using the DFT with positive, negative, and neutral words, Cloitre, Cancienne, Brodsky, Dulit and Perry (1996) tested explicit and implicit memory performance in a group of women with borderline personality disorder (BPD) and a history of parental abuse, a group with BPD but no parental abuse, and a control group (N = 24 in each group). Although the affective valence of the stimuli had to be dropped from the data analyses due to the low numbers of recalled words in each cell, the authors found that the abused BPD group showed an overall greater recall for TBR but no poorer recall for TBF stimuli in the explicit memory condition compared to the other two groups; whereas, no differences were found in the implicit memory performance of the three groups. Cloitre et al. (1996) interpreted these unexpected findings by suggesting that individuals who have been abused in childhood may develop the capacity to focus their attention on designated events (e.g. not associated with the abuse) as a way of coping. Interestingly, memory for TBR but not TBF stimuli was significantly correlated with DES scores, indicating perhaps a propensity for absorption. However, this study did not assess the presence of PTSD symptoms.

Another study (McNally, Metzger, Lasko, Clancy & Pitman, 1998) used the DFT with a group of adult (women) survivor of childhood sexual abuse (CSA) with PTSD (N = 14), a group of women with a history of CSA but no PTSD (N = 12), and a control group (N = 12). As expected, the authors found a standard DFE (i.e. more TBR than TBF words were recalled). However, against predictions, they also found that the PTSD group did not recall fewer TBR or TBF trauma-related than positive or neutral words, but it did exhibit recall deficits for TBR positive and neutral words compared to the other two groups. Therefore, these data were deemed to be inconsistent with the hypothesis of an avoidant encoding or impaired explicit memory for trauma-related information in PTSD. Although the CSA-PTSD group yielded significantly higher scores on the DES, this measure was not related to test performance by the authors.

In a later similar study, McNally et al. (2001) found a standard DFE, but no support for the hypothesis of a specific avoidant encoding for trauma-related information, as tested by free recall, in either a repressed-memory CSA group (N = 13), or a recovered-memory CSA group (N = 13), compared to a control group (N = 15). Once again, the higher DES and trauma measure scores in the CSA groups were not related by the authors to memory performance. DePrince and Freyd (2001) argued that the failure to obtain a differential recall for trauma-related information in McNally et al.'s (1998) study was due to the lack of appreciation for the importance of the attentional context during the DFT. Specifically, based on the findings of their previous study on attention, memory and dissociative tendencies reported above (DePrince & Freyd, 1999), the authors hypothesised that this effect would be found in conditions of divided attention

(i.e. performing the DFT and a parallel simple task at the same time). In fact, in their sample of college students they found that a high-DES group recalled more neutral and fewer trauma-related TBR words compared to a low-DES group ($N = 24$ in each group), in a free recall but not in a recognition task, and only in the condition of divided attention but not selective attention. The authors suggested that dissociation may be adaptive in keeping threatening information from awareness under certain circumstances. Moreover, they proposed that a task requiring divided attention may be more ecologically valid because it approximates more closely the real world, where individuals need to deal frequently with divided attention demands in daily life. However, despite the promising findings reported in this study, participants were college students and no measure of trauma was employed.

More recently, Zoellner et al. (2003) suggested that the failure to support the relationship between avoidant encoding style and PTSD in Cloitre et al.'s (1996) and McNally et al.'s (1998) studies is probably due to the alternation between arousal and avoidant states in this client group, so that, when individuals are in arousal state, an avoidant style may not be detected. To test this hypothesis, the authors used the DFT preceded by either a "serenity" or a "dissociation" mood induction procedure to compare the performance of a PTSD and a control group ($N = 28$ in each group; $N = 14$ once divided by mood induction) in both a free recall and a recognition task. Zoellner et al. (2003) found similar results in the free recall and recognition tasks, with a standard DFE being obtained following the serenity induction, but not following the dissociation induction, which impaired memory for TBR rather than TBF stimuli. Moreover, against their

hypotheses these effects were found in both groups, who also recalled comparable proportions of trauma-related words. The authors explained the elimination of the DFE in the dissociation condition by suggesting that state dissociation may impair the elaboration of incoming information (encoding), and/or may also impair source monitoring (i.e. remember which words were TBR or TBF). Therefore, consistent with previous findings, this study showed no evidence of an avoidant or an intrusive encoding style for trauma-related material in PTSD, although it is very plausible that the mood induction used in this study (i.e. reading a number of relevant phrases such as, "I feel disconnected from my body") may be qualitatively different from dissociation in the real world.

In this regard, two recent studies that have used the DFT in clients with dissociative identity disorder (DID) may help clarify the role of dissociation in conscious and unconscious memory. The first study (Elzinga, de Beurs, Sergeant, van Dyck & Phaf, 2000) compared the performance of a group of individuals with a diagnosis of DID (N = 14), and two groups of university students with high (N = 20) and low (N = 23) dissociation scores, on implicit and explicit memory for sex, threat, and neutral words. The test phase of the DFT was modified in order to separate the two types of memory by using the process dissociation procedure (PDP; Jacoby, 1991). In line with the cognitive avoidance hypothesis, it was expected that instructions to forget would reduce conscious and enhance unconscious memory performance in individuals with high dissociative ability. However, against predictions, the instructions to forget enhanced the overall

(conscious and unconscious) memory performance in the DID group, especially for sex words.

In a second study, Elzinga et al. (2003) investigated directed forgetting within and between personality states of individuals with DID (N = 12) by asking them to switch states between the initial study phase and the test phase, and then back to the initial state for a second test phase. Using this procedure, the authors were able to show a DFE for both emotional (sex/threat) and neutral words between states but not within the same identity state. Thus, although participants were not able to inhibit information selectively within the same state, they appeared to be able to do so when switching to a different state. Moreover, there was a clear reduction in explicit memory performance between states, whereas, implicit memory was largely preserved.

In summary, it would appear that dissociation may have an effect on attentional processes by facilitating dual-tasking and performance under conditions of divided attention (DePrince & Freyd, 1999; 2001), which may also help facilitate selective attention and avoidance in some circumstances (Cloitre et al., 1996), or impair encoding and source monitoring in other situations (Zoellner et al., 2003), although we are still to determine when one or the other might take place.

The effects of dissociation on memory processes are also unclear, in that it may contribute to eliminate the DFE (Zoellner et al., 2003), and even enhance retrieval for trauma-related information (Elzinga et al., 2000), or it may facilitate conscious retrieval inhibition while enhancing unconscious (implicit) memory (Elzinga et al., 2003).

Unfortunately, while some empirical data exist for individuals with BPD, DID and PTSD (although sample sizes are relatively small), our literature searches yielded no study of trauma and dissociation that investigated experimentally attention and memory processes for emotionally laden materials in individuals with psychosis. Therefore our hypotheses regarding the performance on the tasks employed in our study was necessarily based on the studies just reviewed as well as the theoretical background presented earlier in this thesis.

3. 3. Hypotheses.

In order to investigate the potential role played by dissociative processes in the maintenance of psychotic symptoms, as hypothesised above, we decided to examine their relative influence on individuals' recovery style (i.e. level of integration vs. sealing over) from the experience of psychosis, as well as the pathway of information processing for trauma-related, positive, and neutral stimuli in both experimental and control groups.

By doing so we hoped to provide some initial evidence for the presumed negative effects on encoding, storage format, retrieval of information, and level of integration attributed to dissociative processes on theoretical, and some empirical, grounds. Accordingly, our study was set up to provide some preliminary evidence for the existence of a negative interactive system between dissociative processes on the one side and hallucinatory and delusional processes on the other, referred to as CDI in Chapter 2, Section 2. 5.

As mentioned in the opening Section of this thesis, we employed two experimental tasks. The first task was a DFT merged with an emotional Stroop task, referred to hereafter as the Directed Forgetting Stroop Task (DFST), described in detail in the Method Section below. This task was devised in order to provide us with the opportunity to examine attentional processes, to present the study phase for the subsequently administered memory task (see below), and to require participants to perform under conditions of divided attention, which according to the experimental literature reviewed above adds ecological validity and is more likely to involve dissociative processes. Moreover, unlike all of the studies reported above that used the DFT, we used the list rather than the word method. This choice was made based on four premises: *a*) the word method is more likely to tap into encoding processes, which in our case would overlap with the measure of attention we were already taking with the Stroop component of the DFST; *b*) encoding disruption in the word method appears to be due to experimental instructions (i.e. forget) rather than participants' natural selective processes, and as such it might draw on different processes other than dissociation (which might help explain some of the negative research findings in this field); *c*) the list method is more likely to bring into play inhibitory processes and might help reveal the presence of hypothesised different representational formats of memory systems, which might be exposed by the relative proportion of participants' use of implicit and explicit memory; *d*) the results of encoding processes are necessarily reflected in memory functioning because of their temporal precedence.

Following Elzinga et al.'s (2000) approach, during the test phase we applied the process dissociation procedure (PDP; Jacoby, 1991) to a subsequently administered Word-Stem Completion Task (WSCT), in order to discern and estimate the relative contribution of dissociation to implicit and explicit memory processes. The PDP consists of two contrasted conditions: inclusion and exclusion. In the inclusion condition, participants are asked to complete the word-stems with previously presented words as much as possible, and otherwise, with the first word that comes to mind. In this case, explicit (conscious) and implicit (unconscious) memory processes work in the same direction. In contrast, in the exclusion condition, participants are instructed to complete the word-stems only with new words (i.e. not previously seen). This is an interference condition in that if a trial is both consciously controlled and unconsciously influenced, then conscious control and recollection override the unconscious influence and the word-stem is completed with a new word.

Jacoby, Toth and Yonelinas (1993), proposed that conscious memory performance is estimated by subtracting "slip of the tongue" completions in the exclusion condition from the proportion of correctly completed words in the inclusion condition:

$$C = I - E$$

whereas, unconscious memory performance is estimated as:

$$U = E / (1 - C)$$

These formulae were used in our study to estimate conscious and unconscious memory in the WSCT.

We also used four self-report questionnaires and one observer rating scale measuring current severity of psychotic symptomatology, present levels of reported trauma-related symptoms, dissociation, and recovery style. As reported later on in the Method (Section 3. 4.), the Impact of Event Scale – Revised (IES-R; Weiss & Marmar, 1997) was used to assess current levels of trauma-related symptomatology with different referential instructions for the experimental and control groups: the experience of psychosis and the most traumatic/stressful event ever experienced, respectively. This had the aim of gathering a current measure of trauma-related symptomatology in our healthy control group in relation to having experienced as comparable as possible distressing/traumatic life events that might affect their (trauma-related) information processing in our experimental tasks.

In accordance with the background literature reviewed above, it was hypothesised that:

a) during the study phase, people with psychosis would show less interference compared to controls, particularly for trauma-related stimuli, due to their higher levels of dissociation and their presumed more developed ability towards cognitive avoidance in tasks of divided attention;
b) a DFE would be found in both groups (psychosis and control) in their explicit, but not implicit, memory performance;
c) compared to controls, people with psychosis would show a reduced conscious and enhanced unconscious memory performance during the test phase, particularly for trauma-related stimuli, which would indicate conscious retrieval inhibition and facilitation of unconscious activation of information;

d) in people with psychosis, current levels of trauma-related distress would predict the degree of dissociation experienced;

e) in people with psychosis, trauma-related distress and dissociation would predict both their enhanced unconscious memory performance (particularly for trauma-related material) and their recovery style (i.e. degree of sealing over);

f) both enhanced unconscious memory performance and recovery style would predict levels of current positive symptoms in our psychosis group.

3. 4. Method.

3. 4. 1. Experimental design.

The experimental design consisted of a mixed factorial design (3 × 2 × 2 × 2 × 2). There were four within-subjects variables – Word-type (3: trauma-related, positive, categorised neutral); Instruction (2: TBR, TBF); Condition (2: inclusion, exclusion); Stem-type (2: old, new) – and one between-subjects variable – Group (2: psychosis, control).

The dependent variables were: accuracy and reaction times (RTs) in the DFST, completion rates in each experimental condition of the WSCT, and scores from the self-report measures and observer rating scale.

A-priori power analyses (Cohen, 1992; Erdfelder, Faul & Buchner, 1996; Green, 1991; Tabachnick & Fidell, 2001) showed that for multiple regression (with up to 2

independent variables) and ANOVA (repeated measures) designs with $\alpha = .05$, a sample size of $N = 30$ in each group was necessary in order to reveal a large effect size ($f^2 = .35$) at Power = .80; whereas, for t-test analyses a sample size of $N = 26$ in each group was necessary in order to reveal a large effect size ($d = .80$).

3. 4. 2. Participants.

Having obtained ethical approval for the execution of this study from the Lothian Research Ethics Committee (see Appendix 1), two groups of participants were recruited. The first group consisted of 30 individuals (21 males, 9 females; mean age = 40.77, SD = 10.55) who had experienced one or more psychotic episodes in their lives and who were currently well enough to consent and take part in the study. All participants had a diagnosis of Schizophrenia, Paranoid Schizophrenia, Schizoaffective Disorder, or Psychosis. Individuals with a diagnosis of Bipolar Disorder were excluded from this study because, when in euthymic state, they do not usually present any evidence of residual positive symptoms, the maintenance of which is the main topic of this research. Participants in the psychosis (experimental) group were recruited from the Homecare Team, a Community Mental Health Team (CMHT) based in the south-east of Edinburgh. They were all currently engaged with the CMHT and received antipsychotic medication.

In the first instance, potential participants were approached by their keyworker (one of the Community Psychiatric Nurses working within the Team), were given a Patient

Information Sheet (see Appendix 2.), and asked to take part in this study. In doing so, the voluntary nature of participation was emphasised and they were given as long as necessary to decide. On several occasions, I was introduced to individual clients who required further information prior to, or after taking a decision.

The control group consisted of 30 healthy participants (21 males, 9 females; mean age = 40.73, SD = 10.53) with no known history of (or current) emotional disorder who were matched to the psychosis group for age and sex. They consisted of an opportunity sample and included hospital employees, and acquaintances.

3. 4. 3. Apparatus and materials.

Self-report questionnaires. The following self-report questionnaires were used and administered in the given order (see Appendix 3.).

- *Depression Anxiety Stress Scale* (DASS; Lovibond & Lovibond, 1995a). This is a 42-item instrument designed to provide relatively pure measures of current symptoms of depression, anxiety, and stress. It has very good psychometric properties (Cronbach's alphas ranging from .91 to .97, from .81 to .92, and from .89 to .95 for the depression, anxiety, and stress scales respectively), and has been evaluated both in non-clinical and clinical samples (Beuke, Fischer & McDowall, 2003; Brown, Chorpita, Korotitsch & Barlow, 1997; Lovibond, 1998; Lovibond & Lovibond, 1995b). In this study we used the short 21-item

version, which has similar properties and offers advantages for research purposes over the longer version (Antony, Bieling, Cox, Enns & Swinson, 1998).

- *Dissociative Experiences Scale* (DES; Bernstein & Putnam, 1986; Bernstein-Carlson & Putnam, 1993). This is a 28-item measure of dissociative experiences. It is the most widely used measure of dissociation and has shown to have very good psychometric properties (mean Cronbach's α = .93) in several clinical groups (e.g. van Ijzendoorn & Schuengel, 1996). Although this measure contains 3 subscales of dissociation (amnesia, depersonalisation/derealisation, absorption/imaginative involvement), Bernstein-Carlson and Putnam (1993) have claimed that the scale will reliably measure only the general dissociation factor, therefore, we did not use the 3 separate subscales in this study. However, we reported a second score derived from 8 items of the DES, the DES-T (Taxon) which provides a measure of pathological dissociation (Waller et al., 1996).

- *Impact of Event Scale – Revised* (IES-R; Weiss & Marmar, 1997). This is a 22-item questionnaire evaluating experiences of avoidance, intrusion and hyperarousal, which reflect the intensity of post-traumatic phenomena. It is a widely used measure of PTSD symptomatology and it has been shown to have good psychometric properties in clinical and non-clinical samples: Cronbach's α = .87 for avoidance, α = .94 for intrusion, α = .91 for hyperarousal, and α = .91 for the total scale (Creamer, Bell & Failla, 2003).

- *Recovery Style Questionnaire* (RSQ; Drayton et al., 1998). This is a 39-item self-report measure designed as an alternative to the interview measure of recovery style: Integration Sealing Over Scale (ISOS; McGlashan, Wadeson, Carpenter & Levy, 1977). The RSQ requires participants to either agree or disagree with statements about their attitude to their psychotic disorder, and consists of 13 subscales: curiosity, education, optimism, impact, fear, liking, continuity, ownership, responsibility, help-seeking, blame, cause, and satisfaction. These are used to calculate a global score, in percentage, ranging from sealing over to integration, which may also be converted into 1 of 6 categories (e.g. 62-82% is assigned 2 = "tends towards integration"). Although this instrument has not been widely used, Drayton et al. (1998) reported Cronbach's α = .73, and a high correlation with ISOS scores r = .92. In this thesis we used the global dimensional score.

Experimental tasks. The following tasks were employed in this study in the given order.

Directed Forgetting Stroop Task (DFST). This task was a DFT merged with an emotional Stroop task, both of which have been presented above. In this merged version, we included 3 types of word stimuli: trauma-related, positive, and categorised neutral (household items). A total of 120 words (40 of each type) were originally pooled together from materials used in previous published experimental studies (e.g. McNally et al., 1998; Myers, Brewin & Power, 1998) or appositely chosen anew for the present

investigation. Seven independent fellow trainee clinical psychologists then judged each word for its relevance to trauma, for its being positive, and for its being neutral, using a 10-point scale (1 = not at all; 10 = extremely). Mean values were obtained for each word on the three scales and a number of stimuli were discarded because they did not reach cut-off points (i.e. > 7 for the relevant scale, and < 3 for the other two scales). Of the remaining stimuli, 84 words (28 of each type) were chosen and divided into 2 separate lists (A and B) so that each list contained 42 words (14 of each type). The two lists were matched (at a sub-list level) for rating scores, word length, and word frequency determined by Francis and Kucera's (1982) norms (see Table 3. 1., below). Lists A and B were used alternately as TBR or TBF depending on the counterbalancing order (see Procedure, below).

Additionally, 6 more words were used as practice stimuli at the beginning of the task, and further 12 (6 for each list) were used as buffer stimuli at the beginning (3) and the end (3) of the presentation of each list (see Table 3. 2., below).

Furthermore, all the selected word stimuli had a unique stem to be used in the WSCT described below. The DFST was computerised and programmed with the E-Prime Version 1.1 experiment generator package (Schneider, 2003). All the words were presented in uppercase (Bold Ariel size point 30) on a Targa Visionary portable computer (TFT 14" screen). Half of the stimuli in each list (A, B, practice, and buffer) were presented in red ink and half in blue ink on a light grey (silver) background.

Table 3. 1. Lists of word stimuli used for the DFST.

	List A					List B				
Trauma										
1.	Abuse					Aggression				
2.	Ambush					Assault				
3.	Death					Attack				
4.	Harm					Brutal				
5.	Horror					Crime				
6.	Humiliate					Dangerous				
7.	Nightmare					Fear				
8.	Ordeal					Helpless				
9.	Panic					Punishment				
10.	Rape					Shame				
11.	Suffering					Terror				
12.	Threat					Torture				
13.	Traumatic					Victim				
14.	Violent					Weapon				
	T=9.0	*P*=1.1	*N*=1.1	*L*=6.4	*F*=32.3	*T*=8.9	*P*=1.1	*N*=1.3	*L*=6.8	*F*=32.0
Positive										
15.	Affection					Beauty				
16.	Bliss					Comfort				
17.	Caring					Delight				
18.	Cheerful					Divine				
19.	Enjoy					Fantastic				
20.	Genuine					Friendly				
21.	Honest					Happy				
22.	Pleasant					Joke				
23.	Marvellous					Kind				
24.	Positive					Laugh				
25.	Safe					Lovely				
26.	Success					Lucky				
27.	Triumph					Optimist				
28.	Trust					Soothing				
	T=1.2	*P*=8.8	*N*=2.2	*L*=6.8	*F*=33.6	*T*=1.0	*P*=8.5	*N*=2.4	*L*=6.3	*F*=33.6

(*Table continues on the next page*)

Table 3. 1. Lists of word stimuli used for the DFST (*continued*).

	List A					List B				
Neutral										
29.	Cabinet					Chair				
30.	Clock					Curtains				
31.	Fork					Drawer				
32.	Furniture					Freezer				
33.	Kettle					Handle				
34.	Mirror					Iron				
35.	Picture					Ladder				
36.	Porch					Meter				
37.	Stairs					Microwave				
38.	Stool					Radiator				
39.	Suitcase					Scales				
40.	Television					Shelf				
41.	Vase					Table				
42.	Wardrobe					Window				
	T=1.0	*P*=2.1	*N*=9.5	*L*=6.4	*F*=32.1	*T*=1.0	*P*=2.1	*N*=9.4	*L*=6.1	*F*=32.0

Note. *T* = Trauma rating score; *P* = Positive rating score; *N* = Neutral rating score; *L* = Length; *F* = Frequency.

Table 3. 2. List of words used as practice and buffer stimuli for the DFST.

Practice		Buffer	
1.	Arrival	List A – Beginning:	
2.	Edinburgh	1.	Dusty
3.	Festival	2.	Giraffe
4.	Garage	3.	Book
5.	Printer	List A – End:	
6.	Supermarket	4.	Mountain
		5.	Petrol
		6.	Universe
		List B – Beginning:	
		7.	Gathering
		8.	Bicycle
		9.	Minute
		List B – End:	
		10.	Official
		11.	Sledging
		12.	Yesterday

Word-Stem Completion Task (WSCT). For this task, the unique 2 or 3-letter word-stems of the 84 test stimuli used in the DFST were matched with an extra 84 unique word-stems beginning with the same letter and of the same number of letters. Thus, two new lists of word-stems were formed (1 and 2) each containing ½ of the stems from list A and ½ from list B (i.e. 21 "old stems"; 7 for each word-type, from each list) and 42 matched "new stems". Therefore, each list, 1 and 2, contained ½ "old" and ½ "new" word-stems, and each ½ "old" stem list, in turn, included ½ TBR and ½ TBF word-stems. Lists 1 and 2 were randomised in two different orders and presented on paper

with "inclusion" or "exclusion" instructions, as described above in Section 3. 3. (see Appendix 4.).

Observer rating scale. This was usually used at the end of the experimental session.

- *Positive and Negative Syndrome Scale* (PANSS; Kay, Fiszbein & Opler, 1987). This is a widely used 30-item measure of positive, negative, composite (positive - negative) and general psychopathology symptoms. It is designed to be completed by the examiner based on third party information (consultation of current multidisciplinary notes and discussion with keyworkers, in our case), direct observation, and semi-structured interview (on a few occasions it was not possible to carry this out fully for various reasons, in which case we had to rely more on the first two sources). The PANSS has been shown to have good internal consistency and inter-rater reliability: $\alpha = .73$ and $r = .93$ for the positive scale, $\alpha = .83$ and $r = .94$ for the negative scale, and $\alpha = .79$ and $r = .84$ for the general psychopathology scale (Bell, Lysaker, Beam-Goulet, Milstein & Lindenmayer, 1994; Kay et al., 1987). For the purpose of this study, we used scores obtained on the positive, negative and general psychopathology scales.

3. 4. 4. Procedure.

Participants were tested individually with only the experimenter being present in the room, the duration of the session ranging between approximately 1 and 2 hours.

They were initially asked if they had any further questions regarding the study based on the information provided, and then to sign a consent form. The self-report questionnaires were administered in the order given above. The instructions for the IES-R differed for the two groups. Individuals in the psychosis group were instructed to complete the scale in relation to their most recent psychotic episode and hospitalisation, unless participants indicated that a previous episode (e.g. the first one) had been experienced as being more stressful and traumatic. Participants in the control group were instructed to refer to the most traumatic and stressful life event they had ever experienced. The approximate date of the events was recorded for both groups.

Although the control group was not required to disclose the nature of their traumatic experience, a number of people referred to events, such as nearly losing their new born baby, discovering that their long-term partner was having an affair, the sudden death of a parent; whereas, a number of other participants who did not wish to disclose their event, reported a very precise date, indicating that the event was very salient in their lives.

Moreover, the control group was not asked to fill in the RSQ, as this refers specifically to psychosis.

After completing the questionnaires, participants were introduced to the DFST. The portable computer was placed in front of them on a desk so that the screen was

approximately 50 cm away, and they were asked to read the instructions on the screen which were as follows:

"In this task some words will appear one at a time in the middle of the screen preceded by a cross. Some of the words will be presented in RED and some in BLUE. Your task is to PRESS the corresponding coloured key on the keyboard each time AS QUICKLY AS YOU CAN, and also to try and REMEMBER THE WORDS. There will be a short break halfway through. Before we begin there will also be a short practice with a few words so that you can become familiar with the task."

These instructions were then rephrased by the experimenter, who also reassured participants about this not being a memory test as such, and that given the number of words nobody could actually remember them all, so that they were only asked to do their best at remembering as many as they could, since, as one participant put it, "this is not to see how good your memory is, but to see how memory works!". However, these instructions were meant to create a situation of divided attention with competing task demands (i.e. speed of response vs. trying to remember). Participants were also told that a full explanation would be given at the end.

All word stimuli were preceded by a fixation cross at the centre of the screen for 1 second, were randomly presented, and the inter-trial interval (ITI), during which the screen was blank, was 2 seconds. Each word was displayed until the corresponding colour key was pressed. If by mistake the incorrect key was pressed, participants received a feedback (a beep sound) to warn them, but the ITI remained the same and, as participants pressed either key, reaction times were recorded.

After a short practice, when the 6 words listed in Table 3. 2. were presented, there was a pause for further questions, followed by the first part of the task. This involved the presentation of 3 buffer words, then either list A or B, which were counterbalanced across participants, and then 3 more buffer words, to avoid primacy or recency effects. Halfway through the task there was a short break and participants were asked to try and forget the words seen during the first part, "as this will help you remember the words you will see during the second part of the task". During the second part, the remaining list was presented using the same procedure, and participants were asked to remember the words that they saw. In addition to lists A and B, the first and second part of the task were counterbalanced so that half of the participants were asked to forget the words presented during the second part, which "will help you remember the words you saw earlier during the first part of the task".

Following the DFST (study phase) the WSCT (test phase) took place. This also had two parts in order to apply Jacoby's (1991) PDP. During the first part, participants were given either list 1 or 2 (in either of the two different randomised orders) on paper and were instructed to complete as many stems as possible (without minding the correct spelling) with words from the DFST (TBR *and* TBF) and otherwise with the first word that came to mind (inclusion condition). This was followed by the exclusion condition, during which the remaining list of word-stems was presented and participants were instructed to complete as many stems as possible with "new words".

The inclusion and exclusion conditions were counterbalanced across participants, and so were the two versions of list 1 and 2, in order to avoid response biases due to presentation order. No time limit was set for the completion of the WSCT.

In the end, the PANSS was completed and participants were debriefed and thanked for their participation.

3. 5. Results.

3. 5. 1. Data reduction and exploratory analyses.

Data from practice and buffer words used in the DFST were removed from any analyses, and variant spellings of test words were considered correct (e.g. *Abused* for *Abuse*).
Mean RTs, proportions of overall completion rates (old and new word-stems), and proportions of correctly completed old stems, were calculated for each participant in each experimental condition. Moreover, estimates of implicit and explicit memory effects were computed by using the formulae reported above. Boxplots revealed a small number of outliers (3.26%) randomly distributed across the variables of interest (i.e. self-report measures and experimental conditions – including all of the computed variables used in the subsequent analyses). These were dealt with through winsorization, by substituting each outlier with the nearest non-outlier value of the corresponding distribution (Winer, 1971).

Following this procedure, further exploratory data analyses were carried out in order to ascertain whether our data met the assumptions regarding normality, homogeneity of variance and sphericity required for parametric tests. Normality was assessed by examining the skewness and kurtosis of each distribution and by carrying out Kolmogorov-Smirnov's Normality tests. Skewness and kurtosis were deemed to deviate significantly from normality if their values were equal to or exceeded twice the corresponding standard error (i.e. skewness $\geq .86$, and kurtosis ≥ 1.66). Homogeneity of variance and sphericity were assessed by carrying out Levene's test and Mauchly's test respectively. Analyses revealed that some variables did not meet the assumptions referred to above and consequently, where appropriate, standard data transformations were applied in order to reduce the skewness, kurtosis and heterogeneity of variance. Transformations were generally successful in dealing with the violations of the assumptions for parametric tests. Tables reporting details of values before and after transformations for each variable are presented in Appendix 5.

Due to our control group generally reporting very low levels on the self-report measures adopted (particularly on the DASS-Anxiety, DES, IES-R-Hyperarousal, PANSS-Positive and Negative subscales), assumptions of normality and homogeneity of variance were not met for some of the variables of interest. Accordingly, relevant variables were transformed using the method that yielded the best results in terms of distribution and spread of data as indicated by Kolmogorov-Smirnov's Normality test and Levene's test of Homogeneity of variance reported in Table A. 5. 2., Appendix 5. The following variables were transformed: Number of years since trauma, DASS

subscales, DES (Taxon and Total), IES-R (except the Intrusion subscale), and the PANNS-General Psychopathology subscale.

Analyses of the reaction time (RT) data from the DFST showed only a slight positive skew for RTs in the psychosis (positive and neutral words) and control (neutral words) groups (see Table A. 5. 3., Appendix 5.). However, since assumptions required for parametric tests were met as indicated by Kolmogorov-Smirnov's Normality test and Levene's test of Homogeneity of variance, RT variables were not transformed.

On the other hand, data regarding the participants' general task performance on the WSCT (i.e. the proportion of any word-stems completed) were substantially negatively skewed due to both groups performing very well and generating a ceiling effect which was not amenable to any kind of data transformation (see Table A. 5. 3., Appendix 5.). This data set was not of direct relevance to our hypotheses, but rather it constituted a general task performance check. The ceiling effect was expected and was deemed to be a positive result indicating that the task was carried out with relative ease by both groups. Nonetheless, since normality and homogeneity of variance assumptions were not met, results were analysed by using non-parametric statistical tests.

Exploratory data analyses also indicated that data from 4 of the 12 experimental conditions of the WSCT were not normally distributed in both groups (different conditions in each group), and that for a minority of the variables (including computed totals used in our analyses) Levene's test indicated that the variance was heterogeneous.

As reported in Table A. 5. 5. (Appendix 5), it was decided to apply a natural logarithmic transformation to the data in order to relatively improve the shape of their distribution.

Finally, with regards to the computed estimates of conscious and unconscious memory effects, data largely met assumptions for parametric tests with the exceptions of a minority of the experimental conditions in the control group (see Table A. 5. 6., Appendix 5). Since no transformation appeared to significantly improve data distribution, variables were not transformed.

Throughout our analyses, whenever homogeneity of variance could not be assumed as indicated by Levene's test, adjusted degrees of freedom were used to calculate the *t* values in group comparisons. Similarly, whenever the sphericity assumption required for ANOVA was violated as indicated by Mauchly's test, the relevant degrees of freedom were adjusted to calculate the *F* values by using Greenhouse-Geisser's correction factor (Epsilon). However, in view of the fact that a small minority of variables were not normally distributed across the experimental conditions in either of our two groups, the interpretation of some of our results should proceed with caution.

3. 5. 2. Participant characteristics.

Table 3. 3. Participant characteristics.

Variable	Group		Test	$p <$
	Psychosis (N=30)	Control (N=30)		
Sex (M/F)	21/9	21/9	$\chi^2(1) = 0.00$	1
Age	40.77 (10.55)	40.73 (10.53)	$t(58) = 0.01$	.99
# of years since trauma	3.92 (2.51)	5.82 (4.22)	$t(58) = 1.29$	.20
DASS				
Depression	14.87 (9.45)	3.13 (3.22)	$t(58) = 6.24$	.001
Anxiety	10.87 (8.08)	0.92 (1.57)	$t(58) = 10.04$	.001
Stress	13.73 (8.05)	7.43 (4.60)	$t(58) = 3.26$	.01
DES				
Taxon	14.97 (12.10)	2.63 (3.28)	$t(58) = 6.54$	.001
Total	24.65 (14.39)	7.08 (7.33)	$t(58) = 6.69$	.001
IES-R				
Avoidance	1.66 (.94)	.56 (.55)	$t(58) = 5.65$	.001
Intrusion	1.64 (.98)	.80 (.71)	$t(58) = 3.79$	.001
Hyperarousal	1.38 (1.03)	.27 (.36)	$t(58) = 6.24$	.001
Total	1.57 (.87)	.57 (.56)	$t(58) = 5.51$	.001
RSQ	62.08 (12.49)	N/A	—	—
PANSS				
Positive scale	15.97 (3.39)	7.00 (.00)	$t(29)^a = 14.49$	.001
Negative scale	18.17 (5.11)	7.00 (.00)	$t(29)^a = 11.96$	.001
Gen. Ψpathology	48.60 (7.34)	19.77 (2.37)	$t(58) = 25.40$	.001

Note. All the means and standard deviations refer to data cleared of outliers but untransformed in order to aid comprehension and ease comparisons. DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; N/A = Not Applicable; [a] = Equal variances not assumed – adjusted degrees of freedom. Standard Deviations in parentheses.

The two groups did not differ significantly in sex ratio, age, or number of years since the traumatic event referred to in the IES-R. Also, as expected, the control group obtained significantly lower scores than the psychosis group on all the clinical measures (see

Table 3. 3. above). Since after transformation the data for DASS-Anxiety and IES-R-Hyperarousal subscales were still not normally distributed for the control group, we repeated these group comparisons by using non-parametric Mann-Whitney tests. Results remained unchanged for both DASS-Anxiety $U = 47.00$, $p < .001$ and IES-R-Hyperarousal $U = 130.00$, $p < .001$.

3. 5. 3. Experimental tasks.

3. 5. 3. 1. Directed Forgetting Stroop Task.

Participants' accuracy on the DFST, as measured by the frequency of correctly and incorrectly pressed coloured keys, was overall very good. Only 1% of the participants' responses were recorded as incorrect, and these revealed no meaningful pattern across groups or conditions. A summary of the DFST results (mean RTs and standard deviations) indicating attentional resources allocated by the two groups to the three word-types is reported below in Table 3. 4.

Table 3. 4. DFST. Mean reaction times (in milliseconds) for the two groups in each experimental condition (Standard Deviations in parentheses).

	Group	
Word-type	Psychosis	Control
Trauma	945.23 (319.74)	777.47 (258.68)
Positive	927.67 (321.71)	772.53 (247.50)
Neutral	918.50 (320.40)	787.27 (284.12)

A 3 × 2 repeated measures ANOVA was carried out for the RTs data with Word-type (trauma-related, positive, categorised neutral) as within-subjects factor and Group (psychosis, control) as the between-subjects variable. Results indicated the absence of a significant main effect of Word-type $F(1.80,104.60) = 0.64$, *ns* (degrees of freedom Greenhouse-Geisser corrected), and interaction of Word-type × Group $F(1.80,104.60) = 1.59$, *ns* (degrees of freedom Greenhouse-Geisser corrected). Conversely, a significant effect of Group $F(1,58) = 4.09$, $p < 0.05$ was found; however, contrary to our hypothesis, this indicated greater time latencies yielded by the psychosis group in each of the three experimental conditions (see Table 3. 4. above).

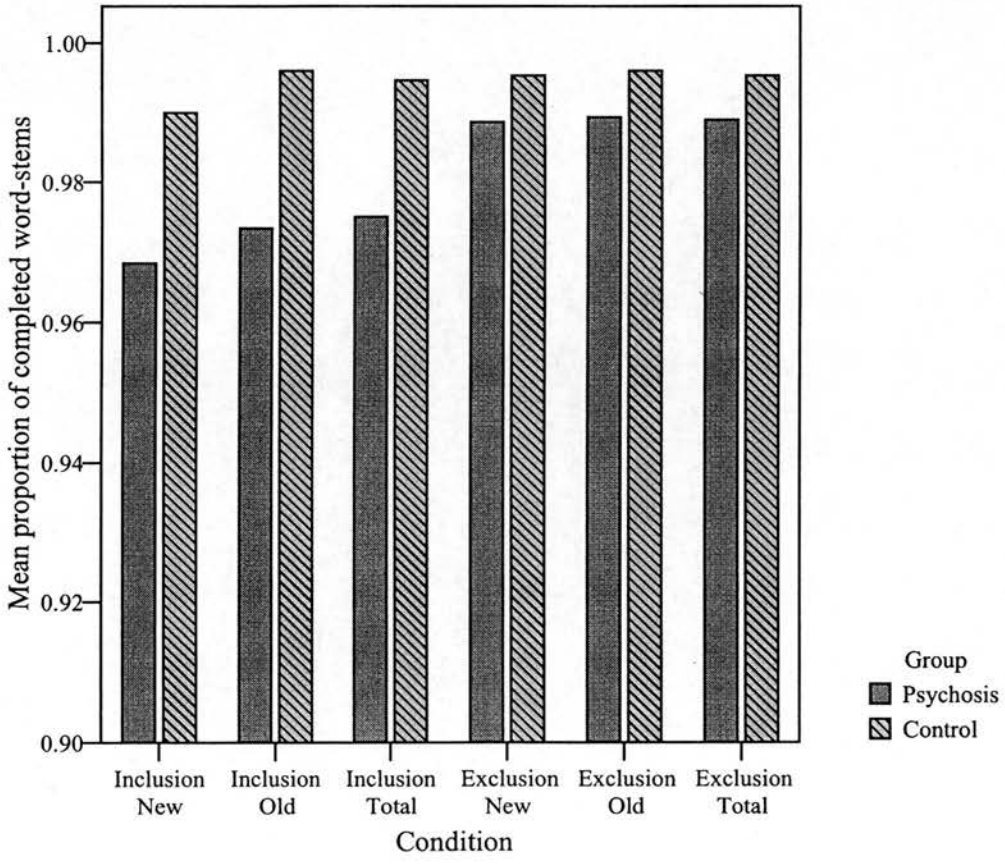
3. 5. 3. 2. Word Stem Completion Task.

The two groups' general task performance on the WSCT, which consists of the proportions of completed old and new stems (with any word) in the inclusion and exclusion conditions, is reported below in Table 3. 5. As already mentioned in Section 3. 5. 1., these data were analysed by using non-parametric statistical tests. Between-groups comparisons showed that, compared to controls, the psychosis group completed relatively smaller proportions of word-stems in the inclusion conditions, significantly so for old stems $U = 288.00$, $p < .01$ and total performance $U = 297.00$, $p < .01$. Within-group comparisons (Wilcoxon tests with Bonferroni's correction) revealed also that the psychosis group completed significantly smaller proportions of word-stems in the inclusion compared to the exclusion conditions for new stems $Z = 2.85$, $p < .05$, old stems $Z = 2.84$, $p < .05$, and totals $Z = 3.14$, $p < .01$ (see Figure 3. 1. below).

Table 3. 5. General performance on WSCT. Mean completion rates of new and old stems for the two groups in each experimental condition (Standard Deviations in parentheses).

		Group			
Condition		Psychosis		Control	
Inclusion:					
	New stems	.968	(.043)	.990	(.016)
	Old stems	.973	(.037)	.996	(.008)
	Total	.975	(.030)	.995	(.008)
Exclusion:					
	New stems	.989	(.015)	.995	(.009)
	Old stems	.989	(.016)	.996	(.008)
	Total	.989	(.015)	.995	(.007)

Figure 3. 1. WSCT. Mean proportions of new and old word-stems completed by the two groups in each experimental condition.



Having analysed the two groups' general task performance, an overarching 2 × 2 × 3 × 2 repeated measures ANOVA was carried out with Condition (inclusion, exclusion), Instruction (TBR, TBF), and Word-type (trauma-related, positive, categorised neutral) as within-subjects variables and Group (psychosis, control) as the between-subjects variable. Table 3. 6., below, shows the descriptive statistics (means and SDs) summarising the performance of the two groups in each of the 12 experimental conditions of the WSCT.

Table 3. 6. WSCT. Mean proportions of correct old stems completions for the two groups in each experimental condition (Standard Deviations in parentheses).

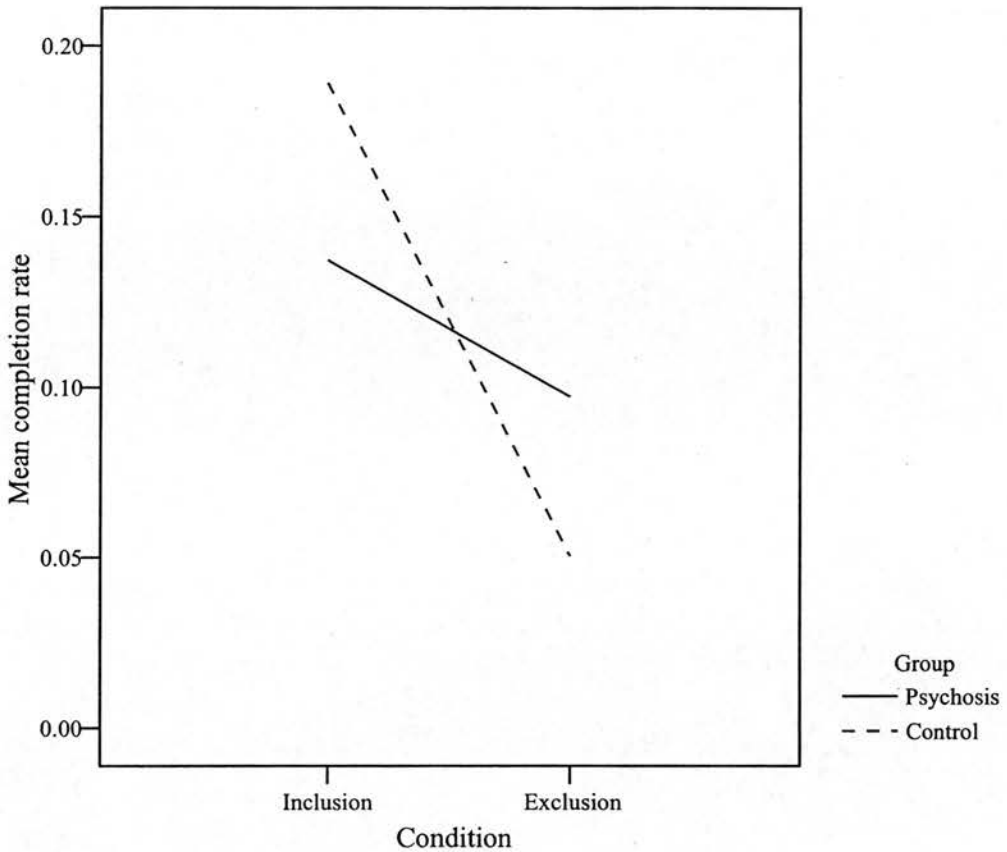
			Group			
Condition			Psychosis		Control	
Inclusion:	TBR					
		Trauma	.163	(.073)	.204	(.099)
		Positive	.151	(.094)	.183	(.085)
		Neutral	.102	(.063)	.178	(.108)
	TBF					
		Trauma	.156	(.104)	.176	(.082)
		Positive	.138	(.089)	.206	(.098)
		Neutral	.106	(.067)	.175	(.096)
Exclusion:	TBR					
		Trauma	.089	(.084)	.035	(.049)
		Positive	.111	(.073)	.064	(.054)
		Neutral	.105	(.078)	.044	(.047)
	TBF					
		Trauma	.091	(.079)	.030	(.048)
		Positive	.099	(.091)	.068	(.076)
		Neutral	.086	(.054)	.058	(.052)

Note. All the means and standard deviations refer to data cleared of outliers before a natural Log-transformation was applied.

As expected, analyses showed a significant main effect of Condition $F(1,58) = 79.99$, $p < 0.001$, reflecting the higher correct completion of old stems in the inclusion compared to the exclusion condition in both groups. There was also a significant main effect of Word-type $F(2,116) = 5.28$, $p < 0.01$, indicating an overall higher completion (with words previously seen) of trauma and positive word-stems compared to neutral ones in both groups. Additionally, we found a significant interaction of Condition × Word-type $F(2,116) = 6.96$, $p < 0.001$, and Condition × Group $F(1,58) = 24.74$, $p < 0.001$. However, the main effect of Instruction was not significant $F(1,58) = 0.45$, *ns*, which indicated that the null hypothesis of no difference in correct completion rates for TBR and TBF word stimuli in both groups could not be rejected. The main effect of Group was also found to be not significant $F(1,58) = 0.008$, *ns*. In order to elucidate the nature of the interactions between Condition × Group, and Condition × Word-type, we performed *post hoc* between and within-groups comparisons. However, given the absence of a significant effect of the variable Instruction, the two experimental conditions TBR and TBF were collapsed.

Between-groups comparisons showed that the significant interaction Condition × Group was due to the psychosis group's significantly lower completion rate in the inclusion condition $t(58) = 3.46$, $p < .001$, and significantly greater completion rate in the exclusion condition $t(46.18) = 4.45$, $p < .001$ (adjusted degrees of freedom), compared to controls (see Figure 3. 2. below). Moreover, within-groups analyses indicated significant differences in completion rates between inclusion and exclusion conditions in both psychosis $t(29) = 2.72$, $p < .01$ and control $t(29) = 10.22$, $p < .001$ groups.

Figure 3. 2. WSCT. Mean proportions of old word-stems completed by the two groups in the inclusion and exclusion conditions.

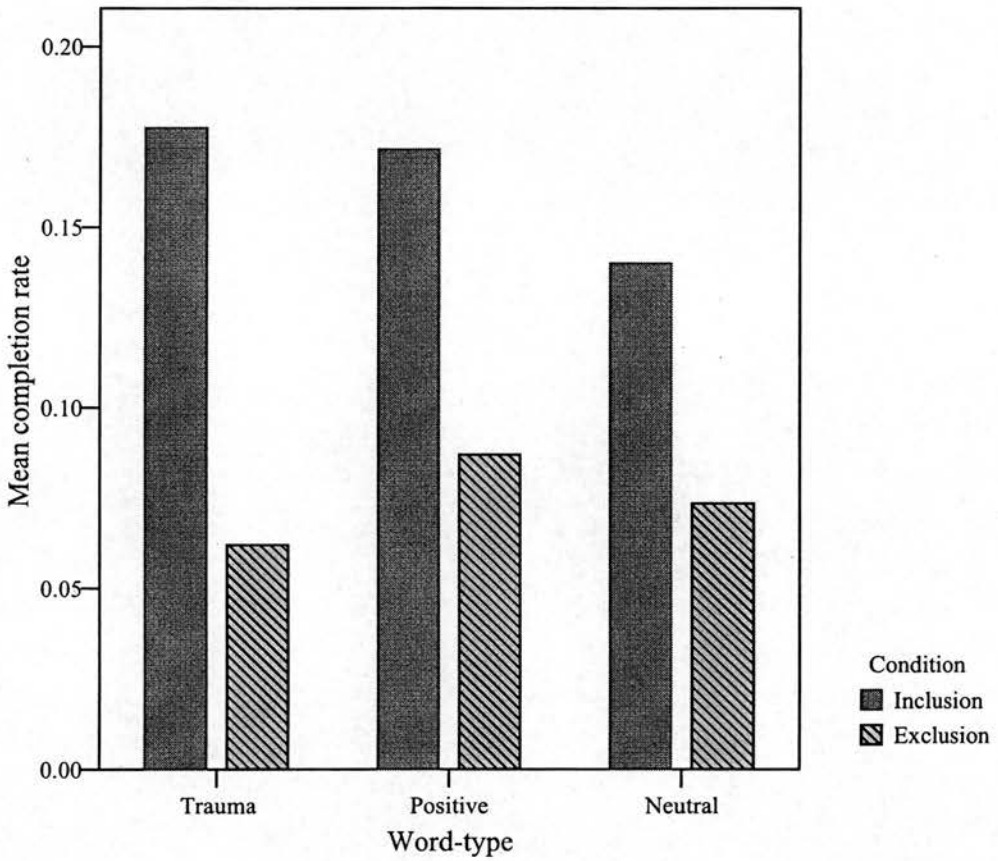


Orthogonal contrasts were carried out for the 6 experimental conditions reported in Figure 3. 3. (below) in order to clarify the interaction of Condition × Word-type.

Of interest, results indicated that participants completed significantly more trauma and positive word-stems than neutral ones in the inclusion condition $F(1,59) = 14.58$, $p < 0.001$, and significantly more positive than trauma word-stems in the exclusion condition $F(1,59) = 10.42$, $p < 0.001$. Moreover, the main effect of Condition reported above was again revealed by significantly higher completion rates for the three word-

types in the inclusion compared to exclusion condition (all significant contrasts $p <$ 0.001).

Figure 3. 3. WSCT. Mean proportions of old trauma, positive, and neutral word-stems completed by the two groups in the inclusion and exclusion conditions.



Having analysed the two groups' performance on the WSCT in terms of the proportions of correctly completed word-stems in all the experimental conditions, we turned to analyse memory effects on the WSCT and carried out an overall 2 × 2 × 3 × 2 repeated measures ANOVA with Memory (conscious, unconscious), Instruction (TBR, TBF), and Word-type (trauma-related, positive, categorised neutral) as within-subjects variables

and Group (psychosis, control) as the between-subjects variable. Table 3. 7., below, shows the descriptive statistics (means and SDs) summarising the conscious and unconscious memory effects for the two groups in each of the 12 experimental conditions of the WSCT. Notably, participants in the psychosis group used slightly more old neutral words to complete word-stems in the exclusion compared to the inclusion condition, as indicated by the negative mean value (-.006) obtained in the Conscious TBR-Neutral experimental condition.

Table 3. 7. WSCT. Mean estimates of conscious and unconscious memory effects for the two groups in each experimental condition (Standard Deviations in parentheses).

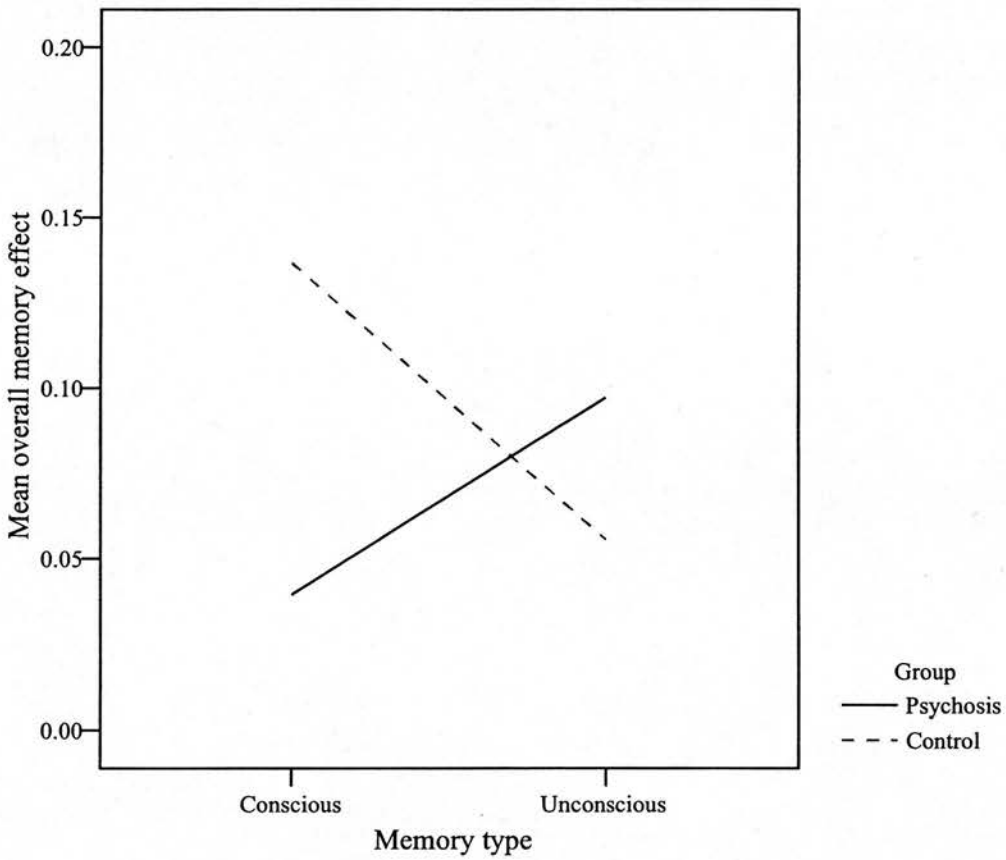
			Group			
Condition			Psychosis		Control	
Conscious:	TBR					
		Trauma	.075	(.108)	.166	(.122)
		Positive	.035	(.112)	.120	(.121)
		Neutral	-.006	(.104)	.133	(.130)
	TBF					
		Trauma	.064	(.141)	.151	(.110)
		Positive	.043	(.147)	.134	(.118)
		Neutral	.025	(.083)	.116	(.112)
Unconscious:	TBR					
		Trauma	.092	(.081)	.041	(.057)
		Positive	.117	(.074)	.072	(.062)
		Neutral	.099	(.071)	.048	(.049)
	TBF					
		Trauma	.092	(.076)	.033	(.052)
		Positive	.097	(.083)	.078	(.088)
		Neutral	.087	(.049)	.064	(.055)

Results showed no main effect of Memory $F(1,58) = 0.65$, *ns*. Also, once again we did not find a significant main effect of Instruction $F(1,58) = 0.23$, *ns*, nor a significant interaction of Memory × Instruction $F(1,58) = 0.15$, *ns*, which indicates the absence of a DFE across memory types, contrary to our experimental hypothesis. However, in line with our hypothesis, analyses revealed a significant main effect of Group $F(1,58) = 12.92$, $p < 0.001$, and interaction Memory × Group $F(1,58) = 23.96$, $p < 0.001$, indicating an overall lower use of conscious memory ($M = .039$, $SD = .084$ vs. $M = .137$, $SD = .077$) and higher use of unconscious memory ($M = .097$, $SD = .046$ vs. $M = .056$, $SD = .029$) in the psychosis group compared to controls. Analyses also showed a significant main effect of Word-type $F(2,116) = 7.07$, $p < 0.001$, and interaction of Memory × Word-type $F(2,116) = 6.07$, $p < 0.01$, reflecting different memory effects across conditions. In order to clarify the interactions between Memory × Group, and Memory × Word-type, we carried out within and between-groups comparisons. However, given the absence of a significant effect of the variable Instruction on explicit and implicit memory, the two experimental conditions TBR and TBF were once again collapsed.

Between-groups comparisons revealed that the significant interaction Memory × Group was due to the psychosis group making significantly less use of conscious memory $t(58) = 4.67$, $p < .001$, and more use of unconscious memory $t(49.23) = 4.15$, $p < .001$ (adjusted degrees of freedom), compared to controls (see Figure 3. 4. below). The estimated effect size for the differential use of conscious memory by the two groups was $d = 1.04$; whereas, for unconscious memory it was $d = .95$. According to Cohen (1992), both these effect sizes can be considered to be large (i.e. $\geq .80$). Additionally, within-

groups analyses indicated significant differences in conscious and unconscious memory effects in both psychosis $t(29) = 2.56$, $p < .05$ and control $t(29) = 4.66$, $p < .001$ groups.

Figure 3. 4. WSCT. Mean overall conscious and unconscious memory effects for the two groups.

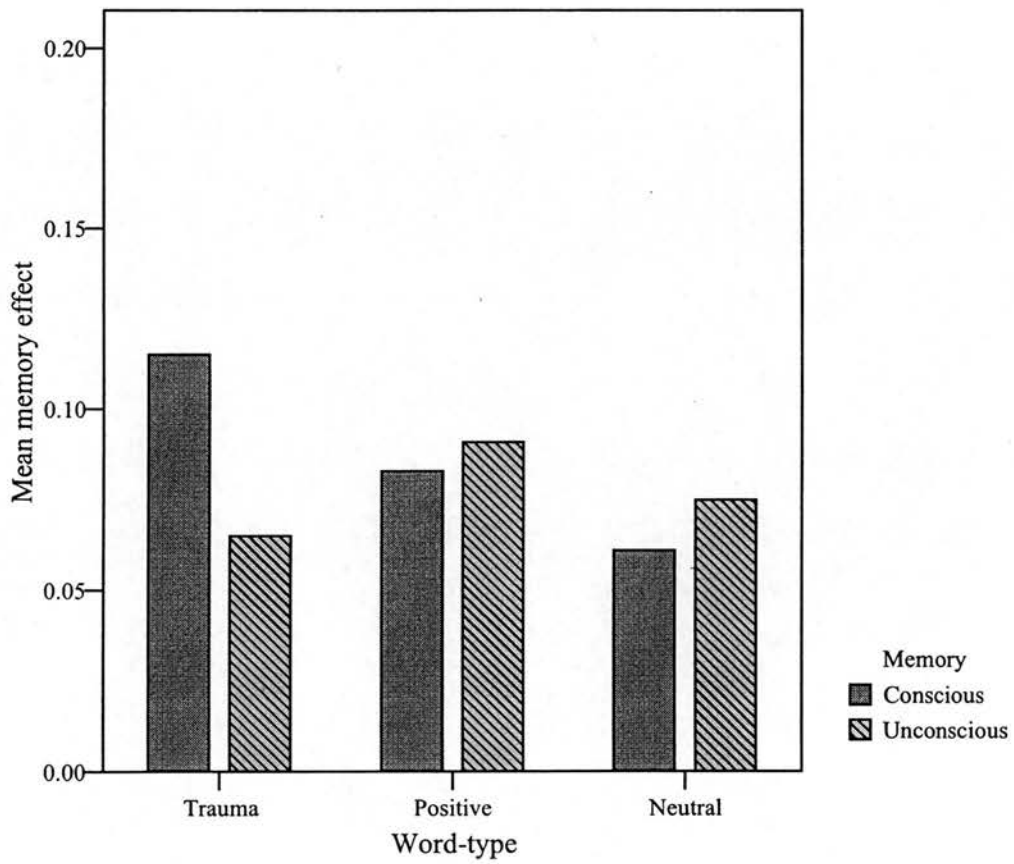


Orthogonal contrasts were carried out for the 6 experimental conditions reported in Figure 3. 5. (below) in order to clarify the interaction of Memory × Word-type.

Of interest, analyses showed that participants made significantly more use of conscious memory for trauma-related words compared to positive $F(1,59) = 5.27$, $p < 0.05$ and

neutral ones $F(1,59) = 20.09$, $p < 0.001$; whereas, they made more use of unconscious memory for positive words compared to trauma-related ones $F(1,59) = 11.24$, $p < 0.001$. Moreover and importantly, the only significant difference in terms of relative usage of conscious and unconscious memory for any given word-type was found to be for trauma-related words $F(1,59) = 5.76$, $p < 0.01$, with a higher proportion of conscious over unconscious memory being employed.

Figure 3. 5. WSCT. Mean conscious and unconscious memory effects for trauma, positive, and neutral word stimuli for the two groups.



3. 5. 4. Correlation and regression analyses.

Zero-order correlation analyses with Bonferroni's correction for multiple tests (see Table 3. 8. below) were performed on the clinical measures adopted in this study so as to explore the relationships between trauma-related distress, dissociation, levels of symptomatology, and recovery style in the psychosis group. As expected Pearson's coefficients (r) revealed a number of significant positive correlations between the measures. Of interest, dissociation (DES) correlated significantly with the Avoidance subscale and Total scores on the IES-R, our measure of trauma-related distress. Moreover, the Stress subscale of the DASS also correlated significantly with the IES-R (Hyperarousal and Total) and the General Psychopathology subscale of the PANSS. Finally, it is worth noting that neither the RSQ nor the PANSS-Positive Scale was significantly correlated to any of the other measures.

In order to test our final hypotheses, we carried out a series of multiple regression analyses (stepwise method) on the data gathered from our psychosis group. Unless otherwise specified, the independent variables entered in each equation were only those measures that according to our hypotheses would be significant predictors for individual factors. However, given the presence of more than two independent variables for some of the regression equations reported below, a note of caution should be mentioned with regards to the possibility that our sample size (N = 30) might be too small to reveal a large effect size (f^2 = .35) at Power = .80 (e.g. Cohen, 1992; Tabachnick & Fidell, 2001).

Table 3. 8. Correlation matrix of clinical measures for the psychosis group ($N = 30$).

		1	2	3	4	5	6	7	8	9	10	11	12	13
1	DASS-D	—												
2	DASS-A	.583	—											
3	DASS-S	.669**	.596*	—										
4	DES-T	.415	.219	.485	—									
5	DES	.457	.316	.522	.742**	—								
6	IES-A	.384	.210	.376	.587	.591*	—							
7	IES-I	.364	.328	.593	.418	.520	.605*	—						
8	IES-H	.583	.507	.806**	.525	.474	.499	.764**	—					
9	IES-T	.474	.388	.636*	.586	.622*	.836**	.911**	.826**	—				
10	RSQ	-.054	-.060	-.024	.253	.478	.241	.285	-.066	.215	—			
11	PANSS-PS	.330	.109	.456	.359	.359	.094	.169	.314	.203	.291	—		
12	PANSS-NS	.169	.073	.159	-.056	.067	-.002	.132	.183	.073	.191	.016	—	
13	PANSS-GP	.340	.390	.600*	.235	.291	.208	.385	.480	.360	.134	.352	.535	—

Note. DASS = Depression Anxiety Stress Scale (D: Depression, A: Anxiety, S: Stress); DES = Dissociative Experiences Scale (T: Taxon); IES-R = Impact of Event Scale – Revised (A: Avoidance, I: Intrusion, H: Hyperarousal, T: Total); RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale (PS: Positive Scale, NS: Negative Scale, GP: General Psychopathology).

* $p < .05$ (2-tailed), Bonferroni's correction.

** $p < .01$ (2-tailed), Bonferroni's correction.

As expected, we found that scores on the DES were significantly predicted by total scores on the IES-R (Adjusted $R^2 = .36$, $F(1,28) = 17.63$, $p < .001$; Standardised $\beta = .62$). However, when the other measures were entered singly into the equation in turn, we also found that scores on the RSQ contributed significantly to predict DES scores ($\Delta R^2 = .13$, $F(1,27) = 6.89$, $p < .01$; Standardised $\beta = .36$). Within this second equation model, IES-R's Standardised $\beta = .54$ ($p < .001$). No further variable added singly to the equation contributed significantly to predict DES scores. Moreover, we found that DES-T scores were significantly predicted by scores on the IES-R-Avoidance subscale (Adjusted $R^2 = .32$, $F(1,28) = 14.71$, $p < .001$; Standardised $\beta = .59$). When the other measures were entered singly into the equation in turn, we found that scores on the PANSS-Positive scale also contributed significantly to predict DES-T scores ($\Delta R^2 = .09$, $F(1,27) = 4.49$, $p < .05$; Standardised $\beta = .31$). Within this second equation model, IES-R-Avoidance's Standardised $\beta = .56$ ($p < .001$). No further variable added singly to the equation contributed significantly to predict DES-T scores.

It was also hypothesised that both trauma-related symptomatology and dissociation would predict our experimental group's enhanced unconscious memory performance (particularly for trauma-related material) and their recovery style (i.e. degree of sealing over). We found that only total scores on the IES-R significantly predicted participants' unconscious memory for trauma-related stimuli (Adjusted $R^2 = .13$, $F(1,28) = 5.35$, $p < .05$; Standardised $\beta = .40$); only scores on DES-T significantly predicted unconscious memory for positive words (Adjusted $R^2 = .27$, $F(1,28) = 11.96$, $p < .01$; Standardised $\beta = .55$); only scores on the IES-R-Hyperarousal significantly predicted our group's overall unconscious memory performance (Adjusted $R^2 = .11$, $F(1,28) = 4.44$, $p < .05$;

Standardised β = .37); whereas, no significant predictors were found for unconscious memory for neutral words. No further measures contributed significantly to the equations once they were entered singly in turn with the above significant predictors.

Results also showed that participants' scores on the RSQ were significantly predicted by total scores on the DES (Adjusted R^2 = .20, $F(1,28) = 8.31$, $p < .01$; Standardised β = .49). When the other measures were entered singly into the equation in turn, we also found that scores on the IES-R-Hyperarousal scale contributed significantly to predict RSQ scores (ΔR^2 = .11, $F(1,27) = 4.53$, $p < .05$; Standardised β = -.38). Additionally, when IES-R-Intrusion was also added to the equation, it contributed further to predict RSQ scores (ΔR^2 = .14, $F(1,26) = 6.91$, $p < .01$; Standardised β = .60). Within this third equation model, DES's Standardised β = .54 ($p < .01$), and IES-R-Hyperarousal's Standardised β = -.78 ($p < .01$). No further variable added singly to the equation contributed significantly to predict RSQ scores.

Finally, we had also hypothesised that both enhanced unconscious memory performance and recovery style would predict levels of positive symptoms in our psychosis group. However, this hypothesis was not supported. Instead, only when all the other self-report measures were entered into the equation as independent variables (stepwise method), the DASS-Stress subscale was found to be the only significant predictor of PANSS-Positive scale (Adjusted R^2 = .18, $F(1,28) = 7.36$, $p < .01$; Standardised β = .46). This was also true when DASS-Stress was entered singly.

In order to test the possibility that the effects of enhanced unconscious memory performance and recovery style on positive symptomatology might be mediated by stress levels, we performed a final linear regression analysis with RSQ and unconscious memory (by word-type and total) as independent variables and DASS-Stress as the dependent variable (stepwise method). Results indicated that the overall enhanced unconscious memory performance was the only significant predictor (Adjusted $R^2 = .10$, $F(1,28) = 4.37$, $p < .05$; Standardised $\beta = .37$). This was also true when it was entered singly, thus supporting only in part our *post hoc* hypothesis.

3. 6. Discussion.

The first aim of this study was to investigate the pathway of trauma-related information processing in individuals with psychosis. This was carried out in order to evaluate experimentally some of the cognitive processes underlying the hypothesis that dissociation occurring as a result of trauma (including the experience of psychosis itself), may render individuals vulnerable to experiencing further psychotic symptomatology (e.g. Allen et al., 1997). Specifically, we proposed that dissociative processes may lead to poorly elaborated and contextually integrated trauma-related information processing, poor awareness and voluntary control of stored material, and its unconscious activation.

As part of our model of trauma, dissociation and psychosis (see Figure 2. 1., Chapter 2), we hypothesised that current levels of trauma-related distress in relation to the experience of psychosis would predict the degree of dissociation experienced by participants in our experimental group. This hypothesis was supported by our results in that, as expected, we found that total scores on the IES-R significantly predicted scores on the DES. Moreover, scores on the IES-R-Avoidance and PANSS-Positive scales both significantly predicted levels of pathological dissociation (DES-Taxon scores). These findings provide further support for the notion that dissociative processes tend to occur as a response to subjectively perceived traumatic life events (e.g. Gershuny & Thayer, 1999; Kihlstrom, 2001; van der Kolk et al., 1996), and that the experience of psychosis is traumatic in nature and can result in the development of significant trauma-related symptomatology and dissociation (e.g. Jackson et al., 2004; McGorry et al., 1991; Shaw et al., 2002).

It was further hypothesised that dissociation would manifest in our psychosis sample through the alteration of attentional and memory processes. In particular, compared to controls, we expected to find less interference (i.e. shorter reaction times) in the psychosis group during the study phase (DFST) of our experiment, due to participants' higher levels of dissociation and their presumed more developed ability towards cognitive avoidance in tasks of divided attention (e.g. DePrince & Freyd, 1999; 2001). It was also proposed that this would be the case particularly when responding to trauma-related stimuli. However, against prediction, we found longer RTs in the psychosis group and no main effect of word-type, which indicate the absence of an advantage of

higher dissociation in the ability to perform when dual-tasking. On balance, the only other study that found this facilitatory effect involved a non-clinical sample of high vs. low dissociation college students (DePrince & Freyd, 1999); whereas, our clinical sample reported severe symptomatology that may well counteract any potential advantage (not to mention the side effects of antipsychotic medication). In fact, motor retardation and generally slower RTs have been found in previous studies of individuals with psychosis (Goldberg, et al., 2003). On the other hand, the absence of a word-type effect might indicate that participants may have deemed necessary to sacrifice speed of performance for the benefit of improving somewhat their memory performance. After all, participants were unaware of the fact that time latencies were being recorded, but they were aware of the fact that their memory for the lists of words appearing on the screen would have been tested, somehow, at the end of the DFST. Consequently, this last finding probably constitutes a methodological limitation of this study, in that we attempted to test attentional processes by using a procedure that emphasised memory functioning. As a result, it is possible that this may have obscured any selective attentional processes (e.g. for trauma-related information) that might have otherwise occurred at the encoding stage. Therefore, although we reject our hypothesis on the basis of the findings obtained here, our study may be inconclusive with respect to the existence of a facilitatory effect of dissociation when performing under conditions of divided attention, a process that may come to light by adopting a different methodological procedure.

With regards to the effects of dissociative processes on memory functioning, one of the main experimental hypotheses was that individuals with psychosis would exhibit a reduced conscious and enhanced unconscious memory performance when completing the word-stems during the WSCT. It was further proposed that this effect would be particularly evident for trauma-related stimuli, which would indicate retrieval inhibition and potential increase in vulnerability due to the increased unconscious de-contextualised activation of trauma-related memories. The results of this investigation supported the first part of this hypothesis, in that, the psychosis group made an overall greater use of unconscious rather than conscious memory compared to the control group, which showed the opposite pattern throughout the task and across conditions. However, the enhanced use of implicit compared to explicit memory in the psychosis group was not specifically larger for trauma-related material, but was generalised also to positive and neutral items (see Table 3. 7., above). Instead, across both groups of participants, we observed more conscious retrieval exclusively for trauma-related material, and more unconscious memory for positive word stimuli.

Thus, our study provides support for the hypothesis that individuals with psychosis tend to process any type of incoming information in a way that facilitates unconscious rather than conscious retention, which may render them vulnerable to its activation without awareness and voluntary control following external stimuli (i.e. in our case: high completion of word-stems with old words in the exclusion condition), and it is also concordant with Hemsley's (1996) cognitive model of schizophrenia which proposes that individuals with psychosis exhibit a weakened ability to integrate information within a temporal and spatial context.

Since individuals with psychosis tend to encode information preferentially implicitly rather than explicitly, the use of standard cognitive techniques (e.g. eliciting evidence, modifying the distressing appraisal of beliefs) may not be as effective as perhaps behavioural experiments, which may be more successful in accessing and modifying emotional responses at both associative and schematic levels (cf. Power & Dalgleish, 1997). Moreover, clients may be unable to consciously access much of the content covered during a session to attempt to implement it in-between sessions, hence the need for repetition and written/audio materials.

Whilst our findings are consistent with previous studies of directed forgetting in PTSD and DID clients, which found enhanced implicit but not explicit memory performance for both emotional and neutral materials (Elzinga et al., 2003), and no retrieval inhibition or better explicit memory for trauma-related material (e.g. Elzinga et al., 2000; McNally et al., 1998; 2001; Zoellner et al., 2003), they might also highlight another possible limitation of our study: the lack of ecological validity for our trauma-related stimuli. Our experiment was obviously not set up to provide participants with a surrogate traumatic experience, but rather to examine cognitive processes particularly in relation to trauma-related information. Moreover, we did not take into account the subjective appraisal of the trauma-related stimuli presented in the DFST, despite their assessed relevance to trauma through a controlled procedure. One of the participants in the experimental group commented that images might have been more effective than words in eliciting emotional responses. Indeed, this is a very valid point and a change in methodology in this direction would certainly add ecological validity to future studies. Therefore, despite the content specificity found for trauma-related stimuli consciously

recalled by participants across both groups, the generalisability of our findings to the way people may process events that are subjectively perceived as traumatic should proceed with caution.

Nonetheless, our hypothesis that, in people with psychosis, enhanced unconscious memory performance (particularly for trauma-related material) would be significantly predicted by levels of trauma-related distress and dissociation was largely supported. In fact, total scores on the IES-R significantly predicted the use of unconscious memory for trauma-related words, the DES-Taxon predicted the use of unconscious memory for positive words, and the Hyperarousal subscale of the IES-R predicted our group's overall enhanced unconscious memory performance. Therefore, taken together, these findings provide support for a strong relationship between trauma and dissociation and their involvement in individuals' cognitive processing styles. However, given the lack of specificity for trauma-related content found in our experimental group's implicit memory performance, the applicability of Brewin et al.'s (1996) and Ehlers and Clark's (2000) models of PTSD in psychosis is inconclusive.

We had also hypothesised that a DFE would be found in both groups in their explicit, but not implicit, memory performance. However, analyses of the estimates of conscious and unconscious memory effects revealed the absence of a DFE, in that, equivalent correct completion rates for TBR and TBF stimuli were found in both groups regardless of explicit or implicit memory contributions. It is possible that this finding may be due to the methodological procedure employed in our study. In fact, in the standard version

of the DFT, the test phase usually composes of a free recall; whereas, we employed a WSCT with contrasting instructions asking participants to use, and not to use, the material previously seen, following Jacoby's (1991) PDP in order to obtain estimates of conscious and unconscious memory effects. Indeed, under these circumstances, a WSCT is equivalent to a forced cued-recall in that participants are encouraged to complete as many stems as possible. In this sense, task demands may act in the same direction as those for a recognition task by facilitating retrieval of primed material (TBR *and* TBF). In support of this interpretation, Basden et al. (1993) found that when the list method of the DFT is used, a recognition test (but not a free recall) reinstates equivalent recall for TBR and TBF items, due to the recall inhibition being released. Arguably, a DFE might not be found in future studies with a psychotic sample even when adopting a standard version of the DFT, because the implicit memory effect in this client group is larger than the explicit memory effect. In fact, it could be possible to observe an inverted DFE (i.e. people with psychosis may remember more TBF than TBR stimuli).

Since in our investigation we used verbal stimuli, according to Brewin et al.'s (1996) model, these should have been dealt with and represented into a VAM system, having received conscious processing. Thus, the higher use of unconscious memory exhibited by the psychosis group in our study would indicate automatic retrieval of verbally accessible information following the presentation of word-stems (a classic example of priming and implicit memory). However, our task was not a typical task attempting to assess implicit memory, since participants were aware of the nature of the task from the beginning, and they were also given clear instructions which made explicit reference to

the use (inclusion), or not (exclusion), of the material previously studied. As a result, controls used predominantly conscious memory to perform in the task. So the question remains as to what may have led our psychosis group to use preferentially unconscious rather than conscious memory.

Our results showed that, compared to controls, the psychosis group completed significantly more word-stems (with old words) in the exclusion condition and less word-stems in the inclusion condition. Therefore, whilst controls were largely able to correctly decide the origin of words used in the WSCT (i.e. presented during the DFST, or just come up to mind), our experimental group exhibited source monitoring deficits. Source monitoring, a deficit of which is a well known phenomenon in this client group (e.g. Brebion, Smith, Gorman & Amador, 1996; 1997; Brebion, Amador, David, Malaspina, Sharif & Gorman, 2000; Brebion, Gorman, Amador, Malaspina & Sharif, 2002; Nienow & Docherty, 2004), refers to those processes involved in making attributions about the origins of memories, knowledge and beliefs (e.g. Johnson, Hashtroudi & Lindsay, 1993). Source monitoring decisions are based on certain characteristics of the memories being judged, such as perceptual, contextual, and affective information, strength (familiarity) of memories, and amount of cognitive operations that were established during encoding of the memory traces. Thus, the more distinct and the greater the amount of the above characteristics, the more accurate decisions will be.

Given that source monitoring deficits imply that memory for imagined and perceived events have similar phenomenological characteristics and entail the blurring of the

boundaries between imagination and reality, our psychosis group's memory performance on the WSCT appears to have been the result of either a dimmed external reality and an abnormally salient internal reality, or a disruption of the comparison process between internal and external events. Anyhow, whichever the mechanism, it becomes apparent that the end result resembles closely some of the effects of dissociation described earlier in this thesis (e.g. derealisation, detachment, absorption). Consequently, we believe that dissociation is largely responsible for such a deficit in our sample. In fact, as we have seen above, dissociation disrupts two key elements of consciousness: awareness and voluntary control, both of which would be needed to perform well according to the instructions in our task. This proposal is also supported by the finding that dissociation was a significant predictor of the use of unconscious memory in the psychosis group.

Accordingly, our results suggest that dissociation contributed significantly to our psychosis sample processing verbal information (supposedly stored within a VAM system in a verbal format) in a way that resembles the format that characterises representations in the SAM system. However, since Brewin et al.'s (1996) model predicts that it is only information represented in the SAM system that can receive no or little conscious attention, lacks context, is poorly elaborated and can be patchy and fragmented, this model cannot explain our findings, in that it does not account for the potential effects of dissociation occurring at different levels of information processing.

A potential alternative candidate may be Kennedy et al.'s (2004) cognitive model of dissociation reviewed in Chapter 2., Section 2. 4. This model proposes that dissociation may occur at different levels: automatic, within-mode, and between-mode, and draws parallels with Brewin et al.'s (1996) model of PTSD. However, given the use of verbal stimuli in our study, and assuming that this information was not dissociated at Stage I (automatically), then, dissociation at Stage II (within-mode) which may result in the severing of associative links among cognitive, behavioural affective and physiological components of events would still not be able to account for our findings. In fact, although the authors have referred to distinct sets of symptomatic manifestations, such as (in the cognitive domain) intrusive thoughts and mind going blank, it is not clear how this could explain what appears to be dissociation happening within (to use their terminology) the cognitive domain itself. This being the case, none of the available models considered can fully accommodate our findings. After all, none of them were developed to account for psychotic experiences, and we would argue for the need of new models specific to psychosis.

The second aim of this thesis was to explore the relationship between severity of symptomatology – particularly current levels of the psychological impact of trauma (i.e. experience of psychosis) and dissociation – and recovery style (i.e. *integration* vs. *sealing over* (e.g. McGlashan, 1987)) in our clinical group. In this regard, our hypothesis of a significant prediction of the psychosis group's recovery style (i.e. degree of sealing over) by the measures of trauma-related distress and dissociation was only partially supported by our findings. In fact, while the prediction of the scores on the IES-R-

Hyperarousal (Standardised β = -.78) and IES-R-Intrusion (Standardised β = .60) subscales went in the anticipated direction, since low scores on the RSQ indicate higher levels of sealing over (i.e. individuals' sealing over recovery style was predicted by an increase in hyperarousal but a decrease in intrusions), the prediction of RSQ by the total scores on the DES (Standardised β = .54) indicated the presence of a relationship between the variables which went in the opposite direction to the one hypothesised (i.e. an increase in sealing over was predicted by a decrease in dissociation levels). These results are consistent with McGlashan's (1987) proposal that sealing over, as a psychological defence, leads to difficulties in accessing memories of one's psychotic episode, and also recent evidence that "sealers", compared to "integrators", report less frequent intrusions as measured by the IES (Jackson et al., 2004). However, contrary to what was hypothesised, our findings show a decrease rather than an increase in dissociation for individuals who seal over.

The significant prediction of higher integration by increasing levels of dissociation may be explained by the fact that individuals who integrate are necessarily trying to deal with their traumatic experience of psychosis (hence the significant prediction of IES-R-Intrusion but not IES-R-Avoidance) and, as such, may be more prone to dissociate in order to buffer the level of distress, at least in the short term. On the other hand, it is likely that the DES as a global measure of dissociation does not fully assess the frequency of dissociative experiences occurring at a tertiary/between modes level, and therefore it is possible that the employment of a more sophisticated measure that is able to discriminate between different levels of dissociation, including their severity, may

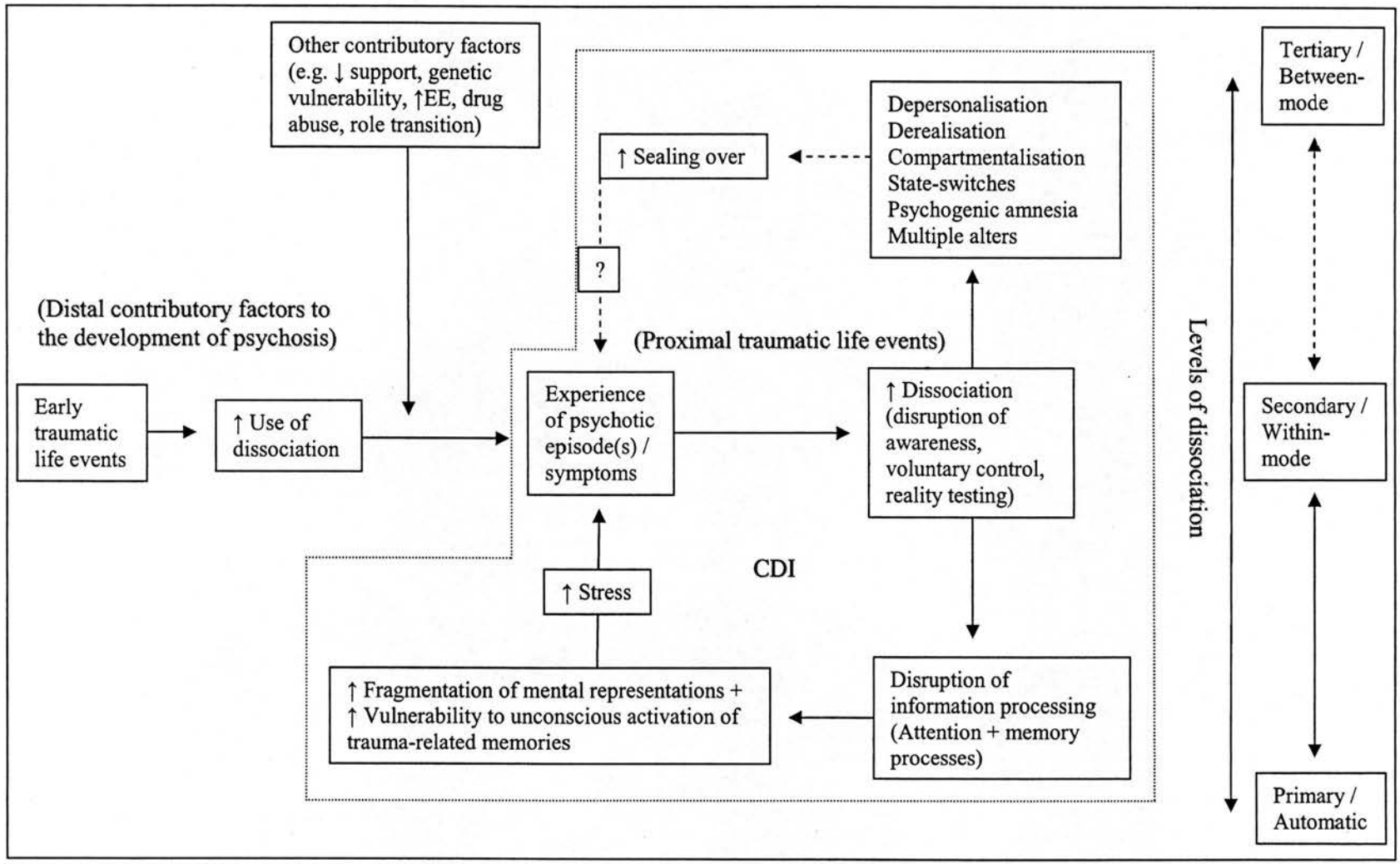
provide a pattern of results that is in line with our hypothesis. To this aim, Kennedy et al. (2004) have sought to develop a new measure (Wessex Dissociation Scale) that is theoretically based on their cognitive model of dissociation, although, to date, this is far from being validated. Arguably, it is also likely that the RSQ may be a better measure of the level of integration than it is of integration vs. sealing over, in that this questionnaire asks individuals to express their views on issues regarding their illness when, according to McGlashan (1987), in individuals who seal over "...psychotic experiences and symptoms are isolated from nonpsychotic mental events and then made unavailable by both conscious suppression and repression" (p. 681). Therefore, a self-report measure does not appear to be the most effective mode of accessing information which has been compartmentalised and made unavailable to conscious awareness.

Given the likely limitations of the RSQ, it is perhaps not surprising that the presence of a linear prediction, proposed in our final hypothesis, of levels of positive symptomatology by both enhanced unconscious memory performance and recovery style was not confirmed by our data. Nonetheless, results indicated that the effect of unconscious memory (but not recovery style) on positive symptoms may be mediated by raised stress levels. Although this finding might be seen as supportive of stress-vulnerability models of psychosis (e.g. Ciompi, 1988; Zubin & Spring, 1977), the vulnerability factor considered in our study is psychological (i.e. the effects of dissociation on information processing) rather than a biological one.

In summary, our findings largely supported the first of the two CDI routes described in Chapter 2, Section 2. 5. (see Figure 3. 6., below, for a revised version of our CDI model). Specifically, dissociation (occurring as a result of the traumatic experience of psychosis) and trauma-related distress predicted our psychosis group's enhanced use of unconscious memory (though not specific to trauma-related information), which, in turn, significantly contributed to the level of positive symptomatology when mediated by stress levels. However, as we have discussed above, the dissociative processes involved in this negative feedback loop appear to include mechanisms from both primary/automatic and secondary/within-mode levels. In contrast, the second CDI route which hypothesised the involvement of tertiary/between-mode dissociation and recovery style in the maintenance of psychotic symptoms was not supported, although this may reflect a limitation of the self-report measures employed in our study.

Thus, a vulnerable individual's attempts at dealing with stressful/traumatic events may result in increased vulnerability and psychopathology (e.g. PTSD, psychosis), since the common processes of PTSD and PTG (e.g. cognitive intrusions, dissociation, hypervigilance) geared towards gaining an understanding of distressing experiences (an innate need according to evolutionary psychology and trauma theorists such as Horowitz, 1997; and Janoff-Bulman, 1992), lead to an exacerbation of symptomatology when acting against a background of cognitive/emotional vulnerability. When an individual's scarce resources (e.g. poor coping skills, lack of social support) are inadequate for him/her to be able to make sense of their psychotic experiences, and incorporate them within his/her existent models of the self and world, he/she may

Figure 3. 6. A cognitive model of trauma, dissociation and psychosis - Revised.



Note. CDI = Cognitive-Defensive Interlock.

dissociate and, potentially, seal over. In this respect the CDI provides an empirically testable framework which, along with existing models of psychosis, may be helpful in formulating some of the basic factors involved in the formation and maintenance of hallucinations and delusions. Of course, more empirical evidence of its existence and *modus operandi* is needed.

3. 7. Conclusions and future directions.

In this thesis we have attempted to address the complex issue of trauma, psychosis, and dissociation. In particular, we have used methods from experimental psychopathology to investigate the potential role played by dissociative processes in the maintenance of psychotic symptomatology. The results of this study have been largely consistent with the experimental hypotheses put forward earlier in this Chapter. Distinctively, the main tenet of this thesis regarding the pivotal role potentially played by dissociative processes in the formation and maintenance of positive symptoms, chiefly hallucinations and delusions, has been supported by the preliminary evidence found in this study concerning the involvement of dissociation in the unique cognitive processing style observed in our psychosis group.

While our choice of methodological procedure and assessment tools might have precluded the positive findings in support of some of our hypotheses, the experimental tasks devised for this study were able to bring to light some of the cognitive processes of

more immediate relevance to the core argument of this thesis (i.e. increased vulnerability in individuals with psychosis due to their unconscious retention and retrieval of information). Nonetheless, given the complexity of individuals' pathways to the development of psychopathology, it would be desirable that future studies assess and include participants' trauma history in order to evaluate its relative contribution to current trauma processing compared to more proximal traumatic life events. As we have seen in our discussion, none of the existing theoretical models considered above can fully account for the enhanced unconscious memory performance observed in our psychosis group, as this appears to have been the result of a combination of dissociative processes acting at different levels of information processing affecting its representational format and subsequent modality of retrieval. Perhaps, the exclusive use of verbal material as stimuli may have been rather restrictive in that, it may have precluded the involvement of lower associative and higher metacognitive levels of mental representations in our investigation. Accordingly, it is recommended that future research into trauma, dissociation, and psychosis examine cognitive processes for complex stimuli (e.g. short vignettes, video clips) of differing emotional valence, coupled with a multilevel test phase devised to tap into the presumed different stages of dissociation and trauma processing, a comprehensive model of which is long awaited.

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Appendices

Appendix 1

LOTHIAN RESEARCH ETHICS COMMITTEE

CERTIFICATE OF ETHICAL OPINION

LREC Reference Number: LREC/2003/7/37
Title: Dissociation and Psychosis: An Experimental Investigation of Attentional and Memory Processes for Trauma-related Information.
Researcher: Dr Massimo Tarsia

The Primary Care/Public & Mental Health Research Ethics Committee of the Lothian Research Ethics Committee (the Committee) reviewed this proposed research and is of the opinion that it is ethical and appropriate to be carried out in the Lothian Area. This opinion encompasses all aspects of the application including the Patient/Subject Information Sheet and all other accompanying documentation provided.

The LREC application form, protocol, subject information sheet, information on compensation arrangements, payments to researchers and the provision of expenses to subjects (where appropriate) were reviewed and approved and the members of the Committee present at the meeting are shown on the attached *Membership List*.

This opinion is issued subject to the following conditions and is invalid if they are not followed:

- You must obtain appropriate management approval from the relevant NHS Trust(s) before starting the proposed research. It is the NHS Trust(s) that ultimately decide whether or not this research should go ahead taking account of the advice of the Local Research Ethics Committee.
- You must notify the Sub-Committee and the relevant NHS Trust(s), in advance, of any significant proposed deviation from the original protocol or application form and obtain approval for any such amendments using the *Amendment Approval Request Form.*
- You must submit reports to the Sub-Committee and the NHS Trust(s) once the study is underway if there are any unusual or unexpected results which raise questions about the safety of the research.
- You must report annually on successes, or difficulties, in recruiting subjects in order to provide useful feedback on perceptions of the study among patients and volunteers using the *Progress Report Form.*
- Where the study is terminated prematurely you must report within fifteen days indicating the reasons for early termination.
- You must submit a final report within three months of the completion of the study using the *Progress Report Form.*
- This opinion does not cover the inclusions of adults with incapacity in any study. Such opinion can only be given by the Multi-Centre Research Ethics Committee for Scotland.

Peter Reith
Secretary
Lothian Research Ethics Committee

08 January 2004

Stephanie Butler
Administrator
Primary Care/Public & Mental Health
Research Ethics Committee

Appendix 2

Title of project: The effect of memory on the way we feel.
Name of researcher: Massimo Tarsia
19/12/2003

NHS Lothian

Information Sheet

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

What is the purpose of this study?

The purpose of this study is to find out how people, who have gone through stressful life events and have experienced a psychotic episode, remember things.

We would be very grateful for your collaboration which will help us understand how what people remember in daily life can affect the way they feel.

Do I have to take part?

No, your participation is entirely voluntary. Should you decide not to take part, your decision will not have any effect on your present or future care.

You have time to think about your decision to participate. If you decide to take part, you will be given this information sheet to keep and will be asked to sign a consent form.

If you decide to take part, you are still free to withdraw at any time and without giving any reason.

What do I have to do?

At the beginning, you will be asked to fill in some questionnaires which look at the way you have been feeling recently.

Then, you will be asked to look at and remember some words appearing one at the time on a small computer screen. Our meeting will last approximately 1 hour.

Results of the research:

This study will run for the first six months of the year 2004. All information will be entirely confidential and only kept for the period of this research. Only properly authorised persons (e.g. your psychiatrist) may have access to this information. Your GP will also be informed of your participation in the study and about the nature of the research.

In due course, it will be possible for you to see the results of this research if you wish to do so (these will not include individual findings, but the main results of the study).

You can contact me, Massimo Tarsia (Tel: 0131. 536 9460), or Mr. Ken Laidlaw (as independent adviser, Tel: 0131. 537 6277) for further information.

Please ask if you have any questions.

Thank you for your help

Appendix 3

DASS

For each of the statements below, please circle the number which best indicates how much the statement applied to you **OVER THE PAST WEEK**. There are no right or wrong answers. Do not spend too much time on any one statement.

	Not at all	Some of the time	A good part of the time	Most of the time
1. I found it hard to wind down.	0	1	2	3
2. I was aware of dryness of my mouth.	0	1	2	3
3. I couldn't seem to experience any positive feeling at all.	0	1	2	3
4. I experienced breathing difficulty (e.g. excessively rapid breathing, breathlessness in the absence of physical exertion).	0	1	2	3
5. I found it difficult to work up the initiative to do things.	0	1	2	3
6. I tended to over-react to situations.	0	1	2	3
7. I experienced trembling (e.g. in the hands).	0	1	2	3
8. I felt that I was using a lot of nervous energy.	0	1	2	3
9. I was worried about situations in which I might panic and make a fool of myself.	0	1	2	3
10. I felt that I had nothing to look forward to.	0	1	2	3
11. I found myself getting agitated.	0	1	2	3
12. I found it difficult to relax.	0	1	2	3
13. I felt down-hearted and blue.	0	1	2	3
14. I was intolerant of anything that kept me from getting on with what I was doing.	0	1	2	3
15. I felt I was close to panic.	0	1	2	3
16. I was unable to become enthusiastic about anything.	0	1	2	3
17. I felt that I wasn't worth much as a person.	0	1	2	3
18. I felt I was rather touchy.	0	1	2	3
19. I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat).	0	1	2	3
20. I felt scared without any good reason.	0	1	2	3
21. I felt that life was meaningless.	0	1	2	3

Patient:
Ward:
Date:

DIRECTIONS: This questionnaire consists of 28 questions about experiences that you may have in your daily life. We are interested in how often you have these experiences. It is important, however, that your answers show how often these experiences happen to you when you were not under the influence of alcohol or drugs. To answer the questions, please indicate how often each experience applies to you by marking the line in the appropriate place, for example:

0% |_____| 100%
Never Always

---

1. Some people have the experience of driving a car and suddenly realising that they do not remember what has happened during all or part of the journey. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

2. Some people find that sometimes they are listening to someone talk and they suddenly realise that they did not hear part or all of what was said. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

3. Some people have the experience of finding themselves in a place and having no idea how they got there. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

4. Some people have the experience of finding themselves dressed in clothes that they don't remember putting on. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

5. Some people have the experience of finding new things among their belongings that they do not remember buying. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

6. Some people sometimes find that they are approached by people that they do not know who call them by another name or insist that they have met them before. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

7. Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something, and they actually see themselves as if they were looking at another person. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

8. Some people are told that they sometimes do not recognise friends or family members. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

9. Some people find that they have no memory for some important events in their lives (for example, a wedding). Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

10. Some people have the experience of being accused of lying when they do not think they have lied. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

11. Some people have the experience of looking in a mirror and not recognising themselves. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

12. Some people sometimes have the experience of feeling that other people, objects and the world around them are not real. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

13. Some people sometimes have the experience of feeling that their body does not seem to belong to them. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

14. Some people have the experience of sometimes remembering a past event so vividly that they feel as if they were reliving that event. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

15. Some people have the experience of not being sure whether things that they remember happening really did happen or whether they just dreamed them. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

16. Some people have the experience of being in a familiar place but fining it strange and unfamiliar. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

17. Some people find that when they are watching television or a film they become so absorbed in the story that they are unaware of other events happening around them. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

18. Some people sometimes find that they become so involved in a fantasy or daydream that it feels as though it were really happening to them. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

19. Some people find that they sometimes are able to ignore pain. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

20. Some people find that they sometimes sit staring off into space, thinking of nothing, and are not aware of the passage of time. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

21. Some people sometimes find that when they are alone they talk out loud to themselves. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

22. Some people find that in one situation they may act so differently compared with another situation that they feel almost as if they were two different people. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

23. Some people sometimes find that in certain situations they are able to do things with amazing ease and spontaneity that would usually be difficult for them (for example, sports, work, social situations, etc.). Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

24. Some people sometimes find that they cannot remember whether they have done something or have just thought about doing that thing (for example, not knowing whether they have actually posted a letter or have just thought about posting it). Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

25. Some people sometimes find evidence that they have done something but cannot remember having done it. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

26. Some people sometimes find writings, drawings or notes among their belongings that they must have done but cannot remember doing. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

27. Some people sometimes find that they hear voices inside their head that tell them to do things or comment on things that they are doing. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

28. Some people sometimes feel as if they are looking at the world through a fog so that people and objects appear far away or unclear. Mark on the line to show what percentage of the time this happens to you.

0% |_____| 100%
Never Always

The Impact of Event Scale - Revised

Below is a list of difficulties people sometimes have after stressful life events. Please read each item, and then indicate how distressing each difficulty has been for you DURING THE PAST SEVEN DAYS with respect to _____, how much were you distressed or bothered by these difficulties?

	Not at all	**A little bit**	**Moderately**	**Quite a bit**	**Extremely**
Any reminder brought back feelings about it	0	1	2	3	4
I had trouble staying asleep	0	1	2	3	4
Other things kept making me think about it	0	1	2	3	4
I felt irritable and angry	0	1	2	3	4
I avoided letting myself get upset when I thought about it or was reminded of it	0	1	2	3	4
I thought about it when I didn't mean to	0	1	2	3	4
I felt as if it hadn't happened or wasn't real	0	1	2	3	4
I stayed away from reminders about it	0	1	2	3	4
Pictures about it popped into my mind	0	1	2	3	4
I was jumpy and easily startled	0	1	2	3	4
I tried not to think about it	0	1	2	3	4
I was aware that I still had a lot of feelings about it, but I didn't deal with them	0	1	2	3	4
My feelings about it were kind of numb	0	1	2	3	4

I found myself acting or feeling as though I was back at that time	0	1	2	3	4
I had trouble falling asleep	0	1	2	3	4
I had waves of strong feelings about it	0	1	2	3	4
I tried to remove it from my memory	0	1	2	3	4
I had trouble concentrating	0	1	2	3	4
Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or a pounding heart	0	1	2	3	4
I had dreams about it	0	1	2	3	4
I felt watchful or on-guard	0	1	2	3	4
I tried not to talk about it	0	1	2	3	4

RSQ

Written below is a list of statements about your illness. Please read them carefully and tick the box to show if you agree or disagree.

	Agree	Disagree
1. There was a gradual build up to me becoming ill.		
2. My illness is not part of my personality.		
3. I am responsible for what I think when I am ill.		
4. I am not interested in my illness.		
5. My illness taught me new things about myself.		
6. I need help to solve the problems caused by my illness.		
7. My illness was caused by my difficulties in coping with life.		
8. I have had a nervous breakdown.		
9. I can see positive aspects to my illness.		
10. My illness has had a strong impact on my life.		
11. I am not frightened of mental illness.		
12. I liked some of the experiences I had when I was ill.		
13. My illness has helped me to find a more satisfying life.		
14. My illness came on suddenly and went suddenly.		
15. My illness is part of me.		
16. I am not responsible for my actions when I am ill.		
17. I am curious about my illness.		
18. I understand myself better because of my illness.		
19. I can manage the problems caused by my illness, alone.		
20. Others are to blame for my illness.		
21. I have had a medical illness.		
22. Nothing good came from my illness.		
23. My illness has had little effect on my life.		
24. I am frightened of mental illness.		
25. I didn't like any of the unusual experiences I had when I was ill.		
26. It's hard to find satisfaction with life following my illness.		
27. My illness came on very suddenly.		
28. My illness is alien to me.		
29. I am responsible for my thoughts and feelings when I am ill.		
30. I don't care about my illness now that I am well.		
31. I want to be the person I was before my illness.		
32. Others can help me solve my problems.		
33. My illness was caused by stress in my life.		
34. I have suffered an emotional breakdown.		
35. Being ill had good parts too.		
36. I'm not really interested in my illness.		
37. I liked some of the unusual experiences I had when I was ill.		
38. My life is more satisfying since my illness.		
39. My attitude to mental illness is better now then before I was ill.		

TWELVE WEEK ASSESSMENT

Date of Assessment:..... Observation Period:.....

POSITIVE AND NEGATIVE SYNDROME SCALE

Circle the appropriate rating for each dimension following the specified clinical interview. Refer to the rating manual for item definitions, description of anchoring points, and scoring procedure.

POSITIVE SCALE

	Abs	Min	Mild	Mod	M/S	Sev	X
Delusions	1	2	3	4	5	6	7
Conceptual Disorganisation	1	2	3	4	5	6	7
Hallucinatory Behaviour	1	2	3	4	5	6	7
Excitement	1	2	3	4	5	6	7
Grandiosity	1	2	3	4	5	6	7
Suspiciousness/ persecution	1	2	3	4	5	6	7
Hostility	1	2	3	4	5	6	7

NEGATIVE SCALE

Blunted Affect	1	2	3	4	5	6	7
Emotional Withdrawal	1	2	3	4	5	6	7
Poor Rapport	1	2	3	4	5	6	7
Passivity/ apathy	1	2	3	4	5	6	7
Abstract Thinking	1	2	3	4	5	6	7
Lack of Sontaneity	1	2	3	4	5	6	7
Stereotyped thinking	1	2	3	4	5	6	7

GENERAL PSYCHPATHOLOGY SCALE

Somatic Concern	1	2	3	4	5	6	7
Anxiety	1	2	3	4	5	6	7
Guilt feelings	1	2	3	4	5	6	7
Tension	1	2	3	4	5	6	7
Mannerisms and posturing	1	2	3	4	5	6	7
Depression	1	2	3	4	5	6	7
Motor retardation	1	2	3	4	5	6	7
Uncooperativeness	1	2	3	4	5	6	7
Unusual thought content	1	2	3	4	5	6	7
Disorientation	1	2	3	4	5	6	7
Poor Attention	1	2	3	4	5	6	7
Lack of judgement and insight	1	2	3	4	5	6	7
Disturbance of volition	1	2	3	4	5	6	7
Poor impulse control	1	2	3	4	5	6	7
Preoccupation	1	2	3	4	5	6	7
Active social avoidance	1	2	3	4	5	6	7

SCALE	TOTAL
Positive	
Negative	
Composite	
General Psychopathology	

Investigators Signature:.....

Appendix 4

WSTC 1-1 (I)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using ANY of the words presented to you earlier** (i.e. either *to-be-remembered* or *to-be-forgotten*), otherwise try to complete them using the first word that comes to your mind.

DEC.....	**HAZ**.....	**TER**.....
VA.....	**SCR**.....	**AGG**.....
FAN.....	**SI**.....	**VIR**.....
DEF.....	**DEA**.....	**SAF**.....
OBS.....	**KIN**.....	**HAP**.....
CRI.....	**HOL**.....	**SQU**.....
AUT.....	**MIC**.....	**LE**.....
TEN.....	**CHA**.....	**BAN**.....
CAL.....	**KNO**.....	**DRI**.....
PAN.....	**TON**.....	**DEL**.....
OPT.....	**ATT**.....	**COL**.....
PER.....	**CHI**.....	**PAR**.....
VE.....	**CHU**.....	**SUF**.....
NU.....	**SUI**.....	**AFF**.....
TRI.....	**AD**.....	**BEA**.....
FOR.....	**ANG**.....	**ACC**.....
HIS.....	**LAC**.....	**TAB**.....
TAR.....	**TOU**.....	**SAL**.....
AB.....	**HOR**.....	**FER**.....
SPE.....	**FIG**.....	**FIL**.....
TRA.....	**CAR**.....	**PUR**.....
HON.....	**MOR**.....	**PAS**.....
EN.....	**EX**.....	**STA**.....
PUN.....	**NI**.....	**LAD**.....
PIC.....	**FEA**.....	**DRA**.....
HAS.....	**LO**.....	**CAB**.....
VIC.....	**KA**.....	**PLE**.....
HAN.....	**SCA**.....	**KE**.....

WSTC 1-1 (E)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using only NEW words** (i.e. words not presented to you earlier at any stage).

DEC.....	**HAZ**.....	**TER**.....
VA.....	**SCR**.....	**AGG**.....
FAN.....	**SI**.....	**VIR**.....
DEF.....	**DEA**.....	**SAF**.....
OBS.....	**KIN**.....	**HAP**.....
CRI.....	**HOL**.....	**SQU**.....
AUT.....	**MIC**.....	**LE**.....
TEN.....	**CHA**.....	**BAN**.....
CAL.....	**KNO**.....	**DRI**.....
PAN.....	**TON**.....	**DEL**.....
OPT.....	**ATT**.....	**COL**.....
PER.....	**CHI**.....	**PAR**.....
VE.....	**CHU**.....	**SUF**.....
NU.....	**SUI**.....	**AFF**.....
TRI.....	**AD**.....	**BEA**.....
FOR.....	**ANG**.....	**ACC**.....
HIS.....	**LAC**.....	**TAB**.....
TAR.....	**TOU**.....	**SAL**.....
AB.....	**HOR**.....	**FER**.....
SPE.....	**FIG**.....	**FIL**.....
TRA.....	**CAR**.....	**PUR**.....
HON.....	**MOR**.....	**PAS**.....
EN.....	**EX**.....	**STA**.....
PUN.....	**NI**.....	**LAD**.....
PIC.....	**FEA**.....	**DRA**.....
HAS.....	**LO**.....	**CAB**.....
VIC.....	**KA**.....	**PLE**.....
HAN.....	**SCA**.....	**KE**.....

WSTC 1-2 (I)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using ANY of the words presented to you earlier** (i.e. either *to-be-remembered* or *to-be-forgotten*), otherwise try to complete them using the first word that comes to your mind.

FIL.....	**SCA**.....	**SUF**.....
TER.....	**TAR**.....	**DEL**.....
CAR.....	**DEA**.....	**KA**.....
HIS.....	**TEN**.....	**ATT**.....
HAS.....	**CHU**.....	**PUR**.....
PAS.....	**PUN**.....	**PIC**.....
NU.....	**KIN**.....	**PER**.....
LO.....	**FER**.....	**SUI**.....
NI.....	**FIG**.....	**PAR**.....
CRI.....	**ACC**.....	**SAF**.....
VA.....	**MIC**.....	**VIC**.....
KE.....	**LAC**.....	**HON**.....
SAL.....	**FEA**.....	**TAB**.....
ANG.....	**HAP**.....	**CAB**.....
FAN.....	**HOR**.....	**TOU**.....
SCR.....	**DRI**.....	**DRA**.....
AB.....	**OBS**.....	**HOL**.....
HAN.....	**EN**.....	**HAZ**.....
BAN.....	**PAN**.....	**COL**.....
PLE.....	**OPT**.....	**AFF**.....
CHI.....	**SI**.....	**DEC**.....
TRA.....	**AGG**.....	**STA**.....
MOR.....	**TRI**.....	**TON**.....
KNO.....	**CAL**.....	**LAD**.....
SPE.....	**DEF**.....	**VIR**.....
BEA.....	**AUT**.....	**FOR**.....
SQU.....	**VE**.....	**CHA**.....
AD.....	**EX**.....	**LE**.....

WSTC 1-2 (E)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using only NEW words** (i.e. words not presented to you earlier at any stage).

FIL.....	**SCA**.....	**SUF**.....
TER.....	**TAR**.....	**DEL**.....
CAR.....	**DEA**.....	**KA**.....
HIS.....	**TEN**.....	**ATT**.....
HAS.....	**CHU**.....	**PUR**.....
PAS.....	**PUN**.....	**PIC**.....
NU.....	**KIN**.....	**PER**.....
LO.....	**FER**.....	**SUI**.....
NI.....	**FIG**.....	**PAR**.....
CRI.....	**ACC**.....	**SAF**.....
VA.....	**MIC**.....	**VIC**.....
KE.....	**LAC**.....	**HON**.....
SAL.....	**FEA**.....	**TAB**.....
ANG.....	**HAP**.....	**CAB**.....
FAN.....	**HOR**.....	**TOU**.....
SCR.....	**DRI**.....	**DRA**.....
AB.....	**OBS**.....	**HOL**.....
HAN.....	**EN**.....	**HAZ**.....
BAN.....	**PAN**.....	**COL**.....
PLE.....	**OPT**.....	**AFF**.....
CHI.....	**SI**.....	**DEC**.....
TRA.....	**AGG**.....	**STA**.....
MOR.....	**TRI**.....	**TON**.....
KNO.....	**CAL**.....	**LAD**.....
SPE.....	**DEF**.....	**VIR**.....
BEA.....	**AUT**.....	**FOR**.....
SQU.....	**VE**.....	**CHA**.....
AD.....	**EX**.....	**LE**.....

WSTC 2-1 (I)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using ANY of the words presented to you earlier** (i.e. either *to-be-remembered* or *to-be-forgotten*), otherwise try to complete them using the first word that comes to your mind.

HEA.....	**JE.....**	**TOR.....**
WAR.....	**SER.....**	**ASS.....**
FRI.....	**SLA.....**	**WOR.....**
DIA.....	**HAR.....**	**SUC.....**
SHO.....	**LAU.....**	**JO.....**
DAN.....	**HUR.....**	**TEA.....**
BRI.....	**RAD.....**	**LI.....**
THI.....	**CUR.....**	**CLE.....**
CLA.....	**LAT.....**	**FLO.....**
RAP.....	**VOL.....**	**DIV.....**
SO.....	**BRU.....**	**DAR.....**
PRE.....	**CIR.....**	**RES.....**
WHI.....	**CUS.....**	**THR.....**
OCC.....	**TEL.....**	**BLI.....**
TRU.....	**APP.....**	**COM.....**
FUR.....	**ART.....**	**BEL.....**
MAN.....	**MEA.....**	**WIN.....**
WRI.....	**TRO.....**	**SMA.....**
AMB.....	**HUM.....**	**FRA.....**
THE.....	**HER.....**	**FUN.....**
VIO.....	**CHE.....**	**SEL.....**
MAR.....	**REL.....**	**POL.....**
GEN.....	**GRA.....**	**STO.....**
SHA.....	**ORD.....**	**MET.....**
POR.....	**HEL.....**	**FRE.....**
IM.....	**LU.....**	**CLO.....**
WEA.....	**MUS.....**	**POS.....**
IR.....	**SHE.....**	**MIR.....**

WSTC 2-1 (E)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using only NEW words** (i.e. words not presented to you earlier at any stage).

HEA.....	**JE**.....	**TOR**.....
WAR.....	**SER**.....	**ASS**.....
FRI.....	**SLA**.....	**WOR**.....
DIA.....	**HAR**.....	**SUC**.....
SHO.....	**LAU**.....	**JO**.....
DAN.....	**HUR**.....	**TEA**.....
BRI.....	**RAD**.....	**LI**.....
THI.....	**CUR**.....	**CLE**.....
CLA.....	**LAT**.....	**FLO**.....
RAP.....	**VOL**.....	**DIV**.....
SO.....	**BRU**.....	**DAR**.....
PRE.....	**CIR**.....	**RES**.....
WHI.....	**CUS**.....	**THR**.....
OCC.....	**TEL**.....	**BLI**.....
TRU.....	**APP**.....	**COM**.....
FUR.....	**ART**.....	**BEL**.....
MAN.....	**MEA**.....	**WIN**.....
WRI.....	**TRO**.....	**SMA**.....
AMB.....	**HUM**.....	**FRA**.....
THE.....	**HER**.....	**FUN**.....
VIO.....	**CHE**.....	**SEL**.....
MAR.....	**REL**.....	**POL**.....
GEN.....	**GRA**.....	**STO**.....
SHA.....	**ORD**.....	**MET**.....
POR.....	**HEL**.....	**FRE**.....
IM.....	**LU**.....	**CLO**.....
WEA.....	**MUS**.....	**POS**.....
IR.....	**SHE**.....	**MIR**.....

WSTC 2-2 (I)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using ANY of the words presented to you earlier** (i.e. either *to-be-remembered* or *to-be-forgotten*), otherwise try to complete them using the first word that comes to your mind.

FUN.....	**SHE**.....	**THR**.....
TOR.....	**WRI**.....	**DIV**.....
CHE.....	**HAR**.....	**MUS**.....
MAN.....	**THI**.....	**BRU**.....
IM.....	**CUS**.....	**SEL**.....
POL.....	**SHA**.....	**POR**.....
OCC.....	**LAU**.....	**PRE**.....
LU.....	**FRA**.....	**TEL**.....
ORD.....	**HER**.....	**RES**.....
DAN.....	**BEL**.....	**SUC**.....
WAR.....	**RAD**.....	**WEA**.....
MIR.....	**MEA**.....	**MAR**.....
SMA.....	**HEL**.....	**WIN**.....
ART.....	**JO**.....	**CLO**.....
FRI.....	**HUM**.....	**TRO**.....
SER.....	**FLO**.....	**FRE**.....
AMB.....	**SHO**.....	**HUR**.....
IR.....	**GEN**.....	**JE**.....
CLE.....	**RAP**.....	**DAR**.....
POS.....	**SO**.....	**BLI**.....
CIR.....	**SLA**.....	**HEA**.....
VIO.....	**ASS**.....	**STO**.....
REL.....	**TRU**.....	**VOL**.....
LAT.....	**CLA**.....	**MET**.....
THE.....	**DIA**.....	**WOR**.....
COM.....	**BRI**.....	**FUR**.....
TEA.....	**WHI**.....	**CUR**.....
APP.....	**GRA**.....	**LI**.....

WSTC 2-2 (E)

Below is a list of word-stems. Each stem can be completed in several ways to make up a complete word. For example, the stem "Pro" could be completed as "Profession", "Prompt", "Provide", "Proud", "Proof" etc. Please try to complete as many of the following stems as possible by **using only NEW words** (i.e. words not presented to you earlier at any stage).

FUN.....	**SHE**.....	**THR**.....
TOR.....	**WRI**.....	**DIV**.....
CHE.....	**HAR**.....	**MUS**.....
MAN.....	**THI**.....	**BRU**.....
IM.....	**CUS**.....	**SEL**.....
POL.....	**SHA**.....	**POR**.....
OCC.....	**LAU**.....	**PRE**.....
LU.....	**FRA**.....	**TEL**.....
ORD.....	**HER**.....	**RES**.....
DAN.....	**BEL**.....	**SUC**.....
WAR.....	**RAD**.....	**WEA**.....
MIR.....	**MEA**.....	**MAR**.....
SMA.....	**HEL**.....	**WIN**.....
ART.....	**JO**.....	**CLO**.....
FRI.....	**HUM**.....	**TRO**.....
SER.....	**FLO**.....	**FRE**.....
AMB.....	**SHO**.....	**HUR**.....
IR.....	**GEN**.....	**JE**.....
CLE.....	**RAP**.....	**DAR**.....
POS.....	**SO**.....	**BLI**.....
CIR.....	**SLA**.....	**HEA**.....
VIO.....	**ASS**.....	**STO**.....
REL.....	**TRU**.....	**VOL**.....
LAT.....	**CLA**.....	**MET**.....
THE.....	**DIA**.....	**WOR**.....
COM.....	**BRI**.....	**FUR**.....
TEA.....	**WHI**.....	**CUR**.....
APP.....	**GRA**.....	**LI**.....

Table A. 5. 1. Exploratory data analyses for self-report measures – before transformations.

Variable	Psychosis Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
Age	40.77 (10.55)	0.22	-0.38	0.59 (30)	.88
# years since trauma	3.92 (2.51)	0.58	-0.12	1.02 (30)	.25
DASS					
Depression	14.87 (9.45)	0.34	-0.80	0.57 (30)	.91
Anxiety	10.87 (8.08)	0.98	0.02	1.31 (30)	.07
Stress	13.73 (8.05)	0.09	-0.77	0.61 (30)	.85
DES					
Taxon	14.97 (12.10)	0.94	-0.20	1.01 (30)	.26
Total	24.65 (14.39)	0.83	0.24	0.68 (30)	.75
IES-R					
Avoidance	1.66 (0.94)	0.32	-0.72	0.74 (30)	.65
Intrusion	1.64 (0.98)	0.48	-0.47	0.61 (30)	.85
Hyperarousal	1.38 (1.03)	0.61	-0.55	0.96 (30)	.32
Total	1.57 (0.87)	0.54	-0.59	0.68 (30)	.75
RSQ	62.08 (12.49)	-0.20	0.59	0.85 (30)	.47
PANSS					
Positive	15.97 (3.39)	0.24	0.11	0.62 (30)	.83
Negative	18.17 (5.11)	0.41	-0.07	0.61 (30)	.86
Gen. Ψpathology	48.60 (7.34)	0.45	0.61	0.68 (30)	.74

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 1. Exploratory data analyses for self-report measures – before transformations (*continued*).

Variable	Control Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
Age	40.73 (10.53)	0.49	0.34	0.59 (30)	.88
# years since trauma	5.82 (4.22)	0.28	-1.33	0.91(30)	.38
DASS					
Depression	3.13 (3.22)	0.82	-0.47	1.30 (30)	.07
Anxiety	0.92 (1.57)	1.50	0.89	2.30 (30)	.001
Stress	7.43 (4.60)	0.15	-1.01	0.76 (30)	.61
DES					
Taxon	2.63 (3.28)	1.21	0.33	1.25 (30)	.09
Total	7.08 (7.33)	1.24	0.04	1.43 (30)	.03
IES-R					
Avoidance	0.56 (0.55)	0.41	-1.44	1.19 (30)	.12
Intrusion	0.80 (0.71)	0.80	-0.20	0.89 (30)	.41
Hyperarousal	0.27 (0.36)	1.24	0.18	1.47 (30)	.03
Total	0.57 (0.56)	1.05	0.41	0.87 (30)	.43
RSQ	N/A	—	—	—	—
PANSS					
Positive	7.00 (0.00)	.[a]	.[a]	.[a]	.[a]
Negative	7.00 (0.00)	.[a]	.[a]	.[a]	.[a]
Gen. Ψpathology	19.77 (2.37)	0.10	-0.51	1.09 (30)	.19

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; N/A = Not Applicable; SD = Standard Deviation; df = Degrees of freedom; .[a] = constant value, statistics cannot be computed.

Table A. 5. 1. Exploratory data analyses for self-report measures – before transformations (*continued*).

Variable	Psychosis and Control Groups	
	Levene's test of Homogeneity of variance (df)	$p <$
Age	0.05 (1,58)	.82
# years since trauma	16.73 (1,58)	.001
DASS		
Depression	26.31 (1,58)	.001
Anxiety	44.76 (1,58)	.001
Stress	8.29 (1,58)	.01
DES		
Taxon	29.91 (1,58)	.001
Total	9.10 (1,58)	.01
IES-R		
Avoidance	9.23 (1,58)	.01
Intrusion	2.73 (1,58)	.10
Hyperarousal	29.83 (1,58)	.001
Total	6.32 (1,58)	.01
RSQ	—	—
PANSS		
Positive	.[a]	.[a]
Negative	.[a]	.[a]
Gen. Ψpathology	16.58 (1,58)	.001

Note. DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; df = Degrees of freedom; .[a] = constant value, statistics cannot be computed.

Table A. 5. 2. Exploratory data analyses for self-report measures – after transformations.

Variable	Psychosis Group					
	Transformation	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
Age	N/A	40.77 (10.55)	0.22	-0.38	0.59 (30)	.88
# years since trauma	LGN	1.45 (0.58)	-0.56	-0.36	0.97 (30)	.31
DASS						
Depression	LGN	2.50 (0.88)	-1.50	2.57	0.84 (30)	.47
Anxiety	LGN	2.23 (0.75)	-0.63	1.20	0.75 (30)	.63
Stress	SQRT	3.65 (1.19)	-0.64	-0.05	0.85 (30)	.47
DES						
Taxon	LGN	2.44 (0.93)	-0.80	0.75	0.55 (30)	.92
Total	SQRT	4.86 (1.44)	0.07	0.11	0.55 (30)	.92
IES-R						
Avoidance	SQRT	1.61 (0.29)	0.03	-0.94	0.67 (30)	.76
Intrusion	N/A	1.64 (0.98)	0.48	-0.47	0.61 (30)	.85
Hyperarousal	INV + REFL	1.49 (0.23)	-0.79	-0.16	0.90 (30)	.39
Total	SQRT	1.58 (0.27)	0.28	-0.83	0.67 (30)	.77
RSQ	N/A	62.08 (12.49)	-0.20	0.59	0.85 (30)	.47
PANSS						
Positive	N/A	15.97 (3.39)	0.24	0.11	0.62 (30)	.83
Negative	N/A	18.17 (5.11)	0.41	-0.07	0.61 (30)	.86
Gen. Ψpathology	LGN	3.87 (0.15)	-0.04	0.33	0.59 (30)	.88

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; N/A = Not Applicable; LGN = Logarithmic; SQRT = Square root; INV = Inverse; REFL = Reflected; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 2. Exploratory data analyses for self-report measures – after transformations (*continued*).

Variable	Control Group					
	Transformation	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
Age	N/A	40.73 (10.53)	0.49	0.34	0.59 (30)	.88
# years since trauma	LGN	1.68 (0.77)	-0.55	-0.86	0.73 (30)	.67
DASS						
Depression	LGN	1.08 (0.88)	-0.08	-1.44	1.23 (30)	.10
Anxiety	LGN	0.41 (0.65)	1.13	-0.47	2.37 (30)	.001
Stress	SQRT	2.78 (0.87)	-0.39	-0.64	0.74 (30)	.64
DES						
Taxon	LGN	0.91 (0.88)	0.40	-1.34	1.01 (30)	.26
Total	SQRT	2.60 (1.17)	0.90	-0.52	1.13 (30)	.16
IES-R						
Avoidance	SQRT	1.23 (0.22)	0.29	-1.55	1.17 (30)	.13
Intrusion	N/A	0.80 (0.71)	0.80	-0.20	0.89 (30)	.41
Hyperarousal	INV + REFL	1.16 (0.19)	0.79	-0.84	1.48 (30)	.02
Total	SQRT	1.24 (0.21)	0.77	-0.22	0.88 (30)	.42
RSQ	N/A	N/A	—	—	—	—
PANSS						
Positive	N/A	7.00 (0.00)	.[a]	.[a]	.[a]	.[a]
Negative	N/A	7.00 (0.00)	.[a]	.[a]	.[a]	.[a]
Gen. Ψpathology	LGN	2.98 (0.12)	-0.14	-0.70	1.16 (30)	.14

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; N/A = Not Applicable; LGN = Logarithmic; SQRT = Square root; INV = Inverse; REFL = Reflected; SD = Standard Deviation; df = Degrees of freedom; .[a] = constant value, statistics cannot be computed.

Table A. 5. 2. Exploratory data analyses for self-report measures – after transformations (*continued*).

Variable	Psychosis and Control Groups		
	Transformation	Levene's test of Homogeneity of variance (df)	$p <$
Age	N/A	0.05 (1,58)	.82
# years since trauma	LGN	3.01 (1,58)	.09
DASS			
Depression	LGN	0.29 (1,58)	.59
Anxiety	LGN	0.01 (1,58)	.91
Stress	SQRT	2.08 (1,58)	.15
DES			
Taxon	LGN	0.22 (1,58)	.64
Total	SQRT	0.69 (1,58)	.41
IES-R			
Avoidance	SQRT	2.85 (1,58)	.10
Intrusion	N/A	2.73 (1,58)	.10
Hyperarousal	INV + REFL	0.63 (1,58)	.43
Total	SQRT	2.41 (1,58)	.13
RSQ	N/A	—	—
PANSS			
Positive	N/A	.[a]	.[a]
Negative	N/A	.[a]	.[a]
Gen. Ψpathology	LGN	0.37 (1,58)	.54

Note. DASS = Depression Anxiety Stress Scale; DES = Dissociative Experiences Scale; IES-R = Impact of Event Scale – Revised; RSQ = Recovery Style Questionnaire; PANSS = Positive and Negative Syndrome Scale; N/A = Not Applicable; LGN = Logarithmic; SQRT = Square root; INV = Inverse; REFL = Reflected; SD = Standard Deviation; df = Degrees of freedom; .[a] = constant value, statistics cannot be computed.

Table A. 5. 3. Exploratory data analyses for DFST (RTs) + WSCT (General Performance + Totals) – no transformations.

Variable	Psychosis Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
DFST (RTs)					
Trauma	945.23 (319.74)	0.80	-0.44	0.80 (30)	.54
Positive	927.67 (321.71)	0.92	0.07	0.83 (30)	.50
Neutral	918.50 (320.40)	0.90	-0.18	0.85 (30)	.46
WSCT (General Performance)					
New stems – I	.968 (.043)	-1.21	0.07	1.49 (30)	.02
New stems – E	.989 (.015)	-0.96	-0.59	2.02 (30)	.001
Old stems – I	.973 (.037)	-1.42	1.12	1.49 (30)	.02
Old stems – E	.989 (.016)	-1.07	-0.42	2.12 (30)	.001
Total – I	.975 (.030)	-0.96	-0.31	1.46 (30)	.03
Total – E	.989 (.015)	-1.09	-0.28	1.82 (30)	.01

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DFST = Directed Forgetting Stroop Task; RTs = Reaction Times; WSCT = Word-Stem Completion Task; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 3. Exploratory data analyses for DFST (RTs) + WSCT (General Performance + Totals) – no transformations (*continued*).

Variable	Control Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
DFST (RTs)					
Trauma	777.47 (258.68)	0.52	-0.44	0.71 (30)	.69
Positive	772.53 (247.50)	0.63	-0.38	0.57 (30)	.91
Neutral	787.27 (284.12)	0.87	0.57	0.60 (30)	.86
WSCT (General Performance)					
New stems – I	.990 (.016)	-1.18	-0.21	2.23 (30)	.001
New stems – E	.995 (.009)	-1.33	-0.26	2.59 (30)	.001
Old stems – I	.996 (.008)	-1.58	0.53	2.68 (30)	.001
Old stems – E	.996 (.008)	-1.58	0.53	2.68 (30)	.001
Total – I	.995 (.008)	-1.07	-0.42	2.12 (30)	.001
Total – E	.995 (.007)	-1.26	0.17	2.22 (30)	.001

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; DFST = Directed Forgetting Stroop Task; RTs = Reaction Times; WSCT = Word-Stem Completion Task; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 3. Exploratory data analyses for DFST (RTs) + WSCT (General Performance + Totals) – no transformations (*continued*).

Variable	Psychosis and Control Groups	
	Levene's test of Homogeneity of variance (df)	$p <$
DFST (RTs)		
Trauma	1.15 (1,58)	.29
Positive	1.27 (1,58)	.26
Neutral	0.38 (1,58)	.54
WSCT (General Performance)		
New stems – I	23.66 (1,58)	.001
New stems – E	17.99 (1,58)	.001
Old stems – I	31.46 (1,58)	.001
Old stems – E	19.98 (1,58)	.001
Total – I	30.09 (1,58)	.001
Total – E	16.12 (1,58)	.001

Note. DFST = Directed Forgetting Stroop Task; RTs = Reaction Times; WSCT = Word-Stem Completion Task; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 4. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – before transformations.

Variable	Psychosis Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 exp. cond. + tot.)					
TBR-Trauma – I	.16 (.07)	0.60	-0.51	1.59 (30)	.01
TBR-Trauma – E	.09 (.08)	0.76	0.26	1.04 (30)	.23
TBF-Trauma – I	.16 (.10)	0.21	-0.70	0.91 (30)	.38
TBF-Trauma – E	.09 (.08)	0.52	-0.28	0.96 (30)	.31
TBR-Positive – I	.15 (.09)	0.12	-0.35	0.85 (30)	.47
TBR-Positive – E	.11 (.07)	0.20	-1.00	1.35 (30)	.05
TBF-Positive – I	.14 (.09)	0.42	-0.49	1.04 (30)	.23
TBF-Positive – E	.10 (.09)	0.51	-0.66	1.07 (30)	.20
TBR-Neutral – I	.10 (.06)	0.84	-0.06	1.97 (30)	.001
TBR-Neutral – E	.10 (.08)	0.13	-1.07	0.95 (30)	.33
TBF-Neutral – I	.11 (.07)	0.23	-0.62	1.30 (30)	.07
TBF-Neutral – E	.09 (.05)	0.52	0.35	1.74 (30)	.01
Total-Trauma – I	.16 (.08)	0.31	0.27	0.78 (30)	.58
Total-Trauma – E	.09 (.07)	0.87	0.62	0.78 (30)	.59
Total-Positive – I	.15 (.07)	0.68	-0.12	0.76 (30)	.61
Total-Positive – E	.11 (.07)	0.46	0.31	0.97 (30)	.30
Total-Neutral – I	.10 (.05)	0.17	-0.45	1.04 (30)	.23
Total-Neutral – E	.10 (.05)	-0.35	-0.67	1.10 (30)	.18
Total – I	.14 (.05)	0.56	-0.10	0.60 (30)	.86
Total – E	.10 (.05)	0.14	-0.55	0.43 (30)	.99

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 4. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – before transformations (*continued*).

Variable	Control Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 exp. cond. + tot.)					
TBR-Trauma – I	.20 (.10)	0.03	-0.32	0.95 (30)	.33
TBR-Trauma – E	.04 (.05)	1.09	0.13	2.01 (30)	.001
TBF-Trauma – I	.18 (.08)	0.21	-1.22	1.11 (30)	.17
TBF-Trauma – E	.03 (.05)	1.32	0.56	2.22 (30)	.001
TBR-Positive – I	.18 (.08)	-0.21	-1.11	1.22 (30)	.10
TBR-Positive – E	.06 (.05)	0.23	-1.11	1.21 (30)	.11
TBF-Positive – I	.21 (.10)	-0.02	-1.06	0.99 (30)	.28
TBF-Positive – E	.07 (.08)	0.85	-0.44	1.36 (30)	.05
TBR-Neutral – I	.18 (.11)	0.51	-1.23	1.14 (30)	.15
TBR-Neutral – E	.04 (.05)	0.59	-0.59	1.62 (30)	.01
TBF-Neutral – I	.17 (.10)	0.43	-0.46	0.95 (30)	.33
TBF-Neutral – E	.06 (.05)	0.29	-1.09	1.28 (30)	.07
Total-Trauma – I	.19 (.09)	0.27	-0.89	0.75 (30)	.63
Total-Trauma – E	.03 (.03)	0.56	-0.75	1.33 (30)	.06
Total-Positive – I	.20 (.07)	0.01	-0.27	0.74 (30)	.65
Total-Positive – E	.07 (.05)	0.53	-0.28	1.11 (30)	.17
Total-Neutral – I	.18 (.09)	0.47	-0.57	0.82 (30)	.51
Total-Neutral – E	.05 (.04)	0.08	-1.01	1.08 (30)	.19
Total – I	.19 (.07)	0.20	-0.30	0.61 (30)	.86
Total – E	.05 (.03)	-0.31	-0.61	0.71 (30)	.70

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 4. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – before transformations (*continued*).

Variable	Psychosis and Control Groups	
	Levene's test of Homogeneity of variance (df)	$p <$
WSCT (12 exp. cond. + tot.)		
TBR-Trauma – I	2.59 (1,58)	.11
TBR-Trauma – E	7.79 (1,58)	.01
TBF-Trauma – I	1.60 (1,58)	.21
TBF-Trauma – E	8.63 (1,58)	.01
TBR-Positive – I	0.03 (1,58)	.87
TBR-Positive – E	5.16 (1,58)	.03
TBF-Positive – I	0.35 (1,58)	.56
TBF-Positive – E	2.31 (1,58)	.13
TBR-Neutral – I	15.44 (1,58)	.001
TBR-Neutral – E	10.71 (1,58)	.01
TBF-Neutral – I	3.57 (1,58)	.06
TBF-Neutral – E	0.02 (1,58)	.88
Total-Trauma – I	1.30 (1,58)	.26
Total-Trauma – E	13.54 (1,58)	.001
Total-Positive – I	0.33 (1,58)	.57
Total-Positive – E	1.16 (1,58)	.29
Total-Neutral – I	7.80 (1,58)	.01
Total-Neutral – E	2.65 (1,58)	.11
Total – I	3.05 (1,58)	.09
Total – E	9.04 (1,58)	.01

Note. WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 5. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – after transformations.

Variable	Psychosis Group					
	Transformation	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 exp. cond. + tot.)						
TBR-Trauma – I	LGN	.15 (.06)	0.50	-0.58	1.55 (30)	.02
TBR-Trauma – E	LGN	.08 (.08)	0.59	-0.15	1.08 (30)	.19
TBF-Trauma – I	LGN	.14 (.09)	0.05	-0.81	0.97 (30)	.30
TBF-Trauma – E	LGN	.08 (.07)	0.38	-0.57	1.00 (30)	.27
TBR-Positive – I	LGN	.14 (.08)	-0.07	-0.45	0.93 (30)	.35
TBR-Positive – E	LGN	.10 (.07)	0.11	-0.97	1.32 (30)	.06
TBF-Positive – I	LGN	.13 (.08)	0.27	-0.57	0.96 (30)	.31
TBF-Positive – E	LGN	.09 (.08)	0.39	-0.88	1.10 (30)	.18
TBR-Neutral – I	LGN	.10 (.06)	0.74	-0.10	1.95 (30)	.00
TBR-Neutral – E	LGN	.10 (.07)	0.03	-1.11	1.01 (30)	.26
TBF-Neutral – I	LGN	.10 (.06)	0.11	-0.64	1.26 (30)	.08
TBF-Neutral – E	LGN	.08 (.05)	0.37	0.24	1.70 (30)	.01
Total-Trauma – I	LGN	.15 (.07)	0.12	0.00	0.85 (30)	.47
Total-Trauma – E	LGN	.08 (.07)	0.71	0.21	0.77 (30)	.59
Total-Positive – I	LGN	.13 (.06)	0.56	-0.33	0.79 (30)	.55
Total-Positive – E	LGN	.10 (.06)	0.29	0.01	0.91 (30)	.38
Total-Neutral – I	LGN	.10 (.04)	0.07	-0.40	1.02 (30)	.25
Total-Neutral – E	LGN	.09 (.05)	-0.43	-0.62	1.15 (30)	.14
Total – I	LGN	.13 (.04)	0.46	-0.22	0.59 (30)	.87
Total – E	LGN	.09 (.05)	0.04	-0.61	0.47 (30)	.98

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; LGN = Logarithmic; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 5. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – after transformations (*continued*).

Variable	Control Group					
	Transformation	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 exp. cond. + tot.)						
TBR-Trauma – I	LGN	.18 (.08)	-0.16	-0.35	0.96 (30)	.32
TBR-Trauma – E	LGN	.03 (.05)	1.03	-0.07	2.02 (30)	.001
TBF-Trauma – I	LGN	.16 (.07)	0.13	-1.23	1.06 (30)	.21
TBF-Trauma – E	LGN	.03 (.04)	1.27	0.37	2.23 (30)	.001
TBR-Positive – I	LGN	.17 (.07)	-0.31	-1.09	1.28 (30)	.07
TBR-Positive – E	LGN	.06 (.05)	0.16	-1.16	1.22 (30)	.10
TBF-Positive – I	LGN	.18 (.08)	-0.14	-1.09	1.08 (30)	.19
TBF-Positive – E	LGN	.06 (.07)	0.76	-0.61	1.39 (30)	.04
TBR-Neutral – I	LGN	.16 (.09)	0.43	-1.32	1.17 (30)	.13
TBR-Neutral – E	LGN	.04 (.04)	0.52	-0.75	1.63 (30)	.01
TBF-Neutral – I	LGN	.16 (.08)	0.27	-0.55	0.89 (30)	.41
TBF-Neutral – E	LGN	.06 (.05)	0.23	-1.16	1.30 (30)	.07
Total-Trauma – I	LGN	.18 (.07)	0.16	-0.99	0.72 (30)	.68
Total-Trauma – E	LGN	.03 (.03)	0.52	-0.84	1.34 (30)	.06
Total-Positive – I	LGN	.18 (.06)	-0.14	-0.35	0.76 (30)	.62
Total-Positive – E	LGN	.06 (.05)	0.43	-0.44	1.10 (30)	.18
Total-Neutral – I	LGN	.16 (.07)	0.34	-0.67	0.74 (30)	.64
Total-Neutral – E	LGN	.05 (.04)	0.02	-1.04	1.11 (30)	.17
Total – I	LGN	.17 (.05)	0.07	-0.39	0.56 (30)	.91
Total – E	LGN	.05 (.03)	-0.36	-0.59	0.71 (30)	.69

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; LGN = Logarithmic; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 5. Exploratory data analyses for WSCT (12 experimental conditions + Totals) – before transformations (*continued*).

Variable	Psychosis and Control Groups		
	Transformation	Levene's test of Homogeneity of variance (df)	$p <$
WSCT (12 exp. cond. + tot.)			
TBR-Trauma – I	LGN	2.62 (1,58)	.11
TBR-Trauma – E	LGN	6.93 (1,58)	.01
TBF-Trauma – I	LGN	1.92 (1,58)	.17
TBF-Trauma – E	LGN	7.49 (1,58)	.01
TBR-Positive – I	LGN	0.04 (1,58)	.85
TBR-Positive – E	LGN	3.87 (1,58)	.05
TBF-Positive – I	LGN	0.16 (1,58)	.69
TBF-Positive – E	LGN	1.98 (1,58)	.16
TBR-Neutral – I	LGN	13.06 (1,58)	.001
TBR-Neutral – E	LGN	9.00 (1,58)	.01
TBF-Neutral – I	LGN	2.42 (1,58)	.13
TBF-Neutral – E	LGN	0.17 (1,58)	.68
Total-Trauma – I	LGN	0.92 (1,58)	.34
Total-Trauma – E	LGN	12.72 (1,58)	.001
Total-Positive – I	LGN	0.18 (1,58)	.67
Total-Positive – E	LGN	0.72 (1,58)	.40
Total-Neutral – I	LGN	6.36 (1,58)	.01
Total-Neutral – E	LGN	2.18 (1,58)	.15
Total – I	LGN	2.41 (1,58)	.13
Total – E	LGN	7.90 (1,58)	.01

Note. WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; I = Inclusion; E = Exclusion; LGN = Logarithmic; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 6. Exploratory data analyses for WSCT (12 memory experimental conditions + Totals) – no transformations.

Variable	Psychosis Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 mem. exp. cond. + tot.)					
TBR-Trauma – C	.08 (.11)	-0.03	-0.81	0.88 (30)	.43
TBF-Trauma – C	.06 (.14)	0.53	-0.44	1.00 (30)	.27
TBR-Positive – C	.04 (.11)	-0.20	-0.25	1.21 (30)	.11
TBF-Positive – C	.04 (.15)	0.10	-1.01	0.76 (30)	.60
TBR-Neutral – C	-.01 (.10)	0.86	0.08	1.25 (30)	.09
TBF-Neutral – C	.03 (.08)	0.06	-0.99	1.02 (30)	.25
TBR-Trauma – U	.09 (.08)	0.49	-0.27	1.12 (30)	.16
TBF-Trauma – U	.09 (.08)	0.33	-0.50	1.03 (30)	.24
TBR-Positive – U	.12 (.07)	0.23	-0.54	1.06 (30)	.21
TBF-Positive – U	.10 (.08)	0.17	-1.24	1.16 (30)	.14
TBR-Neutral – U	.10 (.07)	-0.02	-1.03	0.94 (30)	.34
TBF-Neutral – U	.09 (.05)	0.03	0.07	1.27 (30)	.08
Total-Trauma – C	.07 (.11)	0.46	-0.01	0.54 (30)	.93
Total-Positive – C	.04 (.10)	-0.49	-0.84	0.83 (30)	.50
Total-Neutral – C	.00 (.06)	0.21	-0.85	0.91 (30)	.38
Total-Trauma – U	.09 (.07)	0.66	0.04	0.55 (30)	.92
Total-Positive – U	.11 (.06)	-0.11	-0.36	0.66 (30)	.78
Total-Neutral – U	.09 (.05)	-0.31	-0.26	0.97 (30)	.30
Total – C	.04 (.08)	0.48	-0.47	0.71 (30)	.70
Total – U	.10 (.05)	-0.05	-0.44	0.55 (30)	.92

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; C = Conscious; U = Unconscious; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 6. Exploratory data analyses for WSCT (12 memory experimental conditions + Totals) – no transformations (*continued*).

Variable	Control Group				
	Mean (SD)	Skewness	Kurtosis	Kolmogorov-Smirnov's Normality test Z (df)	$p <$
WSCT (12 mem. exp. cond. + tot.)					
TBR-Trauma – C	.17 (.12)	-0.27	0.19	0.80 (30)	.55
TBF-Trauma – C	.15 (.11)	0.35	-0.25	0.93 (30)	.35
TBR-Positive – C	.12 (.12)	0.03	-0.32	0.76 (30)	.61
TBF-Positive – C	.13 (.12)	-0.19	-0.58	0.77 (30)	.59
TBR-Neutral – C	.13 (.13)	0.29	-1.04	1.20 (30)	.11
TBF-Neutral – C	.12 (.11)	0.72	-0.19	1.41 (30)	.04
TBR-Trauma – U	.04 (.06)	1.15	0.40	2.00 (30)	.001
TBF-Trauma – U	.03 (.05)	1.31	0.60	2.22 (30)	.001
TBR-Positive – U	.07 (.06)	0.42	-0.27	1.15 (30)	.14
TBF-Positive – U	.08 (.09)	1.02	0.63	1.35 (30)	.05
TBR-Neutral – U	.05 (.05)	0.28	-1.38	1.67 (30)	.01
TBF-Neutral – U	.06 (.05)	0.09	-1.31	1.33 (30)	.06
Total-Trauma – C	.16 (.10)	0.15	-1.13	0.92 (30)	.36
Total-Positive – C	.13 (.10)	-0.21	-0.59	0.64 (30)	.81
Total-Neutral – C	.12 (.10)	0.76	-0.40	0.91 (30)	.38
Total-Trauma – U	.04 (.04)	0.50	-0.66	1.36 (30)	.05
Total-Positive – U	.07 (.05)	0.35	-0.33	0.60 (30)	.86
Total-Neutral – U	.06 (.04)	0.07	-0.80	0.82 (30)	.51
Total – C	.14 (.08)	0.36	-0.55	0.58 (30)	.89
Total – U	.06 (.03)	-0.40	-0.45	0.70 (30)	.72

Note. Standard Error of Skewness = 0.43; Standard Error of Kurtosis = 0.83; WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; C = Conscious; U = Unconscious; SD = Standard Deviation; df = Degrees of freedom.

Table A. 5. 6. Exploratory data analyses for WSCT (12 memory experimental conditions + Totals) – no transformations (*continued*).

Variable	Psychosis and Control Groups	
	Levene's test of Homogeneity of variance (df)	$p <$
WSCT (12 mem. exp. cond. + tot.)		
TBR-Trauma – C	0.26 (1,58)	.61
TBF-Trauma – C	1.38 (1,58)	.24
TBR-Positive – C	0.12 (1,58)	.73
TBF-Positive – C	1.87 (1,58)	.18
TBR-Neutral – C	3.90 (1,58)	.05
TBF-Neutral – C	2.74 (1,58)	.10
TBR-Trauma – U	3.10 (1,58)	.08
TBF-Trauma – U	3.39 (1,58)	.07
TBR-Positive – U	1.50 (1,58)	.23
TBF-Positive – U	0.04 (1,58)	.84
TBR-Neutral – U	3.89 (1,58)	.05
TBF-Neutral – U	1.69 (1,58)	.20
Total-Trauma – C	0.26 (1,58)	.61
Total-Positive – C	0.12 (1,58)	.73
Total-Neutral – C	7.81 (1,58)	.01
Total-Trauma – U	10.35 (1,58)	.01
Total-Positive – U	0.40 (1,58)	.53
Total-Neutral – U	0.82 (1,58)	.37
Total – C	0.33 (1,58)	.57
Total – U	5.21 (1,58)	.03

Note. WSCT = Word-Stem Completion Task; TBR = To be remembered; TBF = To be forgotten; C = Conscious; U = Unconscious; SD = Standard Deviation; df = Degrees of freedom.